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Medicine and Surgery

A MANUAL OF SURGERY

The MC Co

A

Manual of Surgery

BY

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IN THREE VOLUMES

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PREFACE

THE object of this work is to give a succinct account of modern surgical pathology, diagnosis, and treatment, and it is intended as a manual for practitioners and students.

Much of merely historical interest has been purposely omitted, partly on account of want of space, and partly because a repetition of what is already in print, but now out of date, is undesirable.

In a work such as this it must necessarily happen that certain rare conditions can only be treated of briefly; but so far as possible, I have embodied the results of sixteen years' experience as a hospital surgeon and teacher.

My best thanks are due to those gentlemen who have so kindly rendered me valuable assistance; to Mr. Donald Gunn who has contributed the chapter in vol. ii. on Injuries of the Eye; to my colleagues, Mr. Tubby, Mr. de Santi, and especially Mr. Paton, who has read the entire MSS., and given me many valuable suggestions; he has also given me great assistance in the selection of illustrations. The original illustrations are from drawings made for me by Miss Booth, Mr. C. H. Freeman, and Mr. G. C. Coltart, and I thank them for the care and skill they have exhibited in their work.

I also desire to offer my cordial thanks to those authors and publishers who have permitted me to make use of their illustrations, the source of which I have acknowledged in the text.

Finally, I must thank Messrs. Macmillan for their liberality in the matter of illustrations, and the courtesy with which they have carried out all my suggestions.

C. S.

4 Harley Street, W.,
1899.

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CHAPTER I

The Degenerations, Atrophy, and Hypertrophy

The Degenerations

A tissue is said to be degenerated when, owing to some change in its structure and composition, its physiological importance is deteriorated, and hence its usefulness in the economy is proportionately diminished. A degeneration properly so-called is a true metamorphosis, the normal tissue undergoing some chemical change, whereby it is converted into something else, *e.g.* muscle into fat. A tissue may, however, be unfiltrated or replaced by some new chemical substance, and itself merely undergo atrophy or some genuine degenerative change; this process is known as an infiltration. Metamorphosis and infiltration are usually associated conditions, and in either case the ultimate physiological degradation of the affected tissue is much the same; but in infiltration the repair may be complete, provided that the infiltrating material becomes absorbed. The degenerations may not only affect tissues normal to the organism, but also new growths and inflammatory effusion.

Causes. — Some of the degenerations occur in various tissues as perfectly normal processes in their life-history; thus fatty degeneration occurs in the epithelium cells of the breast during lactation. The actual change therefore cannot be regarded as any new process, and can only be considered as pathological when it occurs at a time, or in a tissue when, on physiological grounds, we should not expect it. Physiological degeneration is then a normal process in the life-history of the cell. The ultimate fate of all living cells is death; the individual tissues, like the body of which they form a part, have a definite life-history; they reach a

certain state of physiological perfection, enjoy this for a time, and then gradually deteriorate and finally die, to be replaced by new cells. No cell in the body is stable; the enjoyment of vital activity entails the death penalty, but between full physiological vigour and death there is the stage of degeneration or gradual atrophy, and hence the causes of these conditions, when considered from a pathological standpoint, must be sought in some state inducing deficiency of normal nutrition.

Nutrition. — The proper maintenance of nutrition is dependent upon a variety of conditions acting harmoniously for the common benefit.

(1) There must be an adequate supply of blood.

(2) The blood must be of good quality. These two conditions imply the due supply of oxygen and the removal of waste material.

(3) There must be that inherent power in the tissues themselves which enables them to convert to their own uses the nutritive material supplied by the blood. This quality depends in great measure upon —

(4) Connection with a healthy nervous mechanism.

The precise influence of the nervous system in general nutrition is at present unknown, but numerous instances of its primary importance may be easily given. Thus a lesion of a nerve leads to nutritive defects in the parts which it supplies, exhibited clinically by skin affections, ulcerations, and sometimes by sloughing, *e.g.* perforating ulcer of the foot, acute bed-sores, and cystitis from spinal injury. No doubt the action of the nervous system is complex, since it regulates the circulation, sensation, and the functional activity of the part.

(5) The due exercise of function serves to maintain proper nutrition, since it is associated with increased circulation, and hence a constant supply of nutritive material and removal of waste products.

The foregoing being the main factors concerned in healthy nutrition, it is to their diminution or overthrow that we must look for the actual causes of degeneration and atrophy; such may be due to —

(1) A deficient supply of blood, caused by actual anæmia, disease of the vessel walls, or pressure from without.

(2) Deteriorated quality of the blood, including its admixture with certain deleterious substance such as toxines, alcohol, phosphorus, lead, etc.

(3) Impairment of the powers of assimilation, which is probably due in the main to—

(4) Impairment or loss of nervous action, and

(5) Impairment or loss of functional activity.

While it is true that the cause of any degeneration must be sought in some departure from the normal standard of nutrition, the ultimate causes must in any given case be sought in those conditions, constitutional or local, to which such departure is directly due. These causes will be considered under each form of degeneration.

Varieties.—The chief forms of degeneration met with are Fatty, Albuminoid, Calcareous, Mucoid, and Colloid.

FATTY DEGENERATION

In fatty Metamorphosis the albumen of the cells is converted into fat; in fatty Infiltration fat is added to the cells or is deposited between them, but is not made from the albumen of the part; the former only is a true degeneration. The two conditions are usually associated.

Causes.—Fatty metamorphosis is chiefly met with in prolonged fevers, chronic suppuration, and wasting diseases, *e.g.* cancer. Local fatty changes may be induced by arterial obstruction.

Fatty infiltration may result from an excess of fat-forming food, or from deficient oxidation, as in pulmonary disease.

Morbid anatomy.—When a cell is undergoing fatty metamorphosis, dark spots appear in its protoplasm; these gradually increase in size and coalesce, the centre of the oil globule is then seen to be highly refracting and surrounded by a dark margin. Ultimately the cell is practically converted into a fat cell, the whole of the protoplasm having undergone degeneration; the cell subsequently disintegrates (Fig. 1, *e*, *f*). In fatty infiltration the cell contains fat, and its normal protoplasm is simply pushed to one side (Fig. 1, *b*). Fatty infiltration is only of pathological importance when the amount of fat stored up in the cells is large enough to impair their functional activity. A part which is the seat of fatty change is usually increased in bulk and

FIG. 1.—Fatty infiltration and fatty degeneration of liver cells (Ziegler). *a*, *b*, fatty deposit in the cells—infiltration; *c*, *d*, *e*, *f*, fatty degeneration and disintegration of the cells.

weight, but both may be diminished; the consistency is altered, the surface is greasy and fatty in appearance, and the colour is pale yellow.

Surgical importance of fatty degeneration.—Since prolonged fever and suppuration are prominent factors in the production of widespread fatty degeneration, it is of the first importance to cut short these processes whenever possible. But apart from this aspect of the question, fatty changes have an effect upon the tissues with which every surgeon should be familiar.

The heart, if fatty, is unfitted for the administration of anæsthetics, or to meet any shock or strain thrown upon it such as a capital operation entails.

The kidneys are rendered incompetent, and hence may give out after an operation, especially one implicating the genito-urinary tract, in consequence of which reflex irritation of these organs may occur. In all cases of renal inadequacy morphia is a particularly dangerous drug.

Fatty degeneration may occur in the **muscles** of paralysed limbs, in wounded **nerves**, or in **amputation stumps**. In the last the degenerative change is a preparatory step to physiological atrophy; but in the case of a wounded nerve the change may be arrested and a *restitutio ad integrum* brought about by timely suture of the divided ends.

The arteries which may require the attention of the surgeon are very rarely the seat of primary fatty change, which is usually limited to the aorta.

Atrophied bones contain more fat than natural, which fills the widened Haversian spaces. Fatty change is a marked feature of osteo-malacia.

Inflammatory products may become fatty, and are then said to be caseated; when such a process occurs, the inflammation is naturally arrested. Granulation tissue may undergo fatty degeneration, provided anything interferes with its due nutrition.

Any **new growth**, especially such as are inadequately supplied with blood, may undergo fatty change, as is almost constantly seen in glandular cancers.

ALBUMINOID DEGENERATION

Causes.—This form of degeneration is essentially dependent upon chronic suppuration, and is met with in chronic disease of bones and joints, phthisis, congenital and acquired syphilis, and some other conditions.

Morbid anatomy.—The change is specially met with in the liver, spleen, kidneys, and intestines. It begins in the intima of the small arteries, spreads to the capillaries (Fig. 2, *b*, *k*, *i*) but not to the veins, and ultimately affects the adjacent proper cells of the organ. The cells are enlarged and rounded, and the protoplasm is gradually replaced by a glistening albuminoid material, the whole being represented by a homogeneous mass. As the degeneration advances the tissue is converted into a dense, shining, glue-like material.

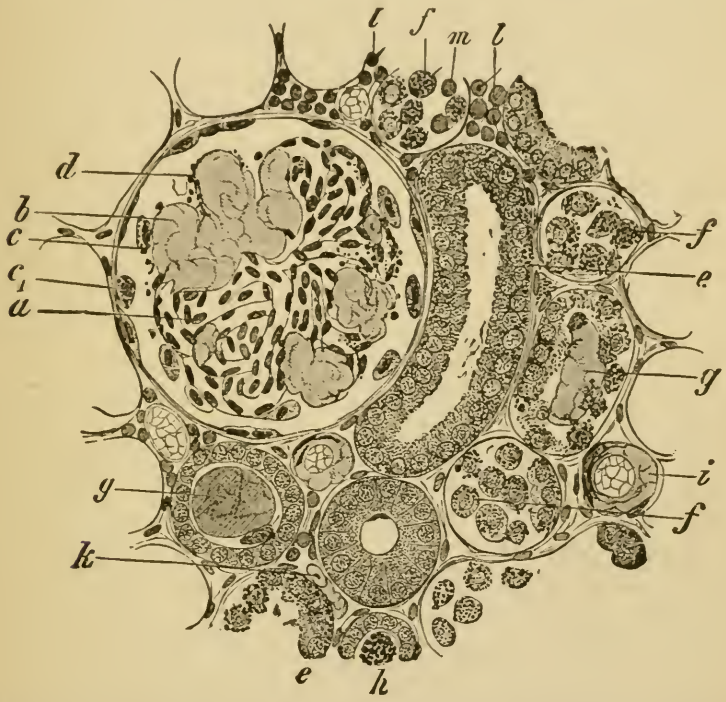

FIG. 2.—Amyloid kidney with fatty degeneration (Ziegler). *a*, capillary; *b* and *k*, amyloid capillary; *c*, *e*, *f*, fatty epithelium; *d*, oil globules; *g*, hyaline casts; *k*, fatty casts; *i*, amyloid arteriole; *l*, cellular infiltration; *m*, round cells within a urinary tubule.

The organ is enlarged, heavy, smooth, elastic, and anæmic in consequence of the narrowing of the blood-vessels by the amyloid matter. Fatty degeneration is almost always associated. The degenerate material is known as lardacein, but is never found in the blood as such; Dickinson regards it as de-alkalised fibrin, resulting from the loss of potassium salts by chronic suppuration.

When treated with an aqueous solution of iodine, albuminoid material is stained a reddish-brown or mahogany colour; but the most delicate test is by methyl-violet, which stains it a brilliant red colour and the unaffected protoplasm an intense blue.

Signs.—An affected organ is uniformly enlarged but painless, and there may be evidence of interference with its functions. When the kidneys are involved there is albuminuria, and in bad cases dropsy or even uræmia may result; diarrhœa is evidence of albuminoid change in the intestines. The general condition of the patient is affected not only by the chronic suppuration, but by the fact of albuminoid degeneration; there is marked anæmia, general debility, and pallor of the skin with cachexia.

Treatment is preventive. Chronic suppuration must be arrested by means applicable to the individual case. In some cases this will effect a marked improvement in the condition, even if the degeneration is advanced; indeed, if the cause be entirely removed, practical recovery may ensue. The diet must be plentiful, and rich in nitrogenous material, with a supply of fruit and green vegetables. The bicarbonate and citrate of potash should be given internally, in view of the great diminution of the potassium salts in the tissues.

COLLOID AND MUCOID DEGENERATION

These conditions are chiefly met with in new growths, but their causes are unknown. The colloid change affects the epithelial cells and is met with in some forms of cancer, especially when affecting the abdominal organs; mucoid degeneration chiefly attacks the cellular tissue. The mucoid and colloid changes are usually associated. Colloid material is jelly-like in appearance and consistency, colourless or pale yellow, and contains albuminates and mucin with sulphur; the last substance is not present in mucin. When the colloid or mucoid change is quite localised, a cyst containing the material is formed. The so-called colloid cancer is merely a cancer which has undergone colloid degeneration.

CALCAREOUS INFILTRATION

Calcification consists in the deposit in the tissues of granules of calcium phosphate with traces of calcium carbonate, and of the phosphate and carbonate of magnesia. The deposit takes place in the connective tissue and the cells.

Causes.—It is probable that there is some antecedent change in the tissues whereby the amount of carbonic dioxide is lessened, and hence the lime salts are precipitated from the fluids; in some cases the change appears to be dependent upon an excess of lime

salts in the blood, *e.g.* osteo-malacia. Calcification is especially prone to affect parts of low vitality, and hence is met with in the aged, in slowly growing new growths, in cyst walls, and in caseous and tubercular foci. When the change is advanced the tissue may be converted into a dense calcareous mass, which has not, however, the anatomical structure of true bone.

In advanced age calcareous changes in the arteries and ligaments are common.

Effects.—A part which has become calcareous may practically be considered as a foreign body, incapable of undergoing any vital change, but sometimes exciting inflammation in its neighbourhood. The effects of calcareous changes in the arteries are fully discussed in chap. i. vol. iii.

Atrophy

Simple atrophy consists in wasting of a tissue without any alteration in its structure; in some cases there is also a diminution in the number of the component elements (Aplasia or Numerical Atrophy).

Causation.—Atrophy is essentially dependent upon failure of nutrition, and consequently is often, in fact usually, associated with degeneration, especially the fatty form. Atrophy differs from degeneration in the fact that there is no chemical change in the cells, and hence, if the cause be removed and nutrition be re-established, complete recovery may occur. Atrophy must be distinguished from congenital arrest of growth. Atrophy occurs as a perfectly normal process in advancing age; in the sexual organs at the climacteric, and in the thymus gland in early life. General atrophy may be merely a senile change, the preparatory step to death, or it may be dependent upon some pathological condition inducing widespread failure of nutrition. Local atrophy is due to some local circulatory disturbance, to functional disuse, to constant pressure, or to interference with the nerve supply of a part. Pressure induces atrophy by lessening the blood supply and diminishing functional activity.

Disuse favours atrophy partly because the tissue is deprived of that periodic flux of blood necessary to healthy nutrition; paralysed limbs waste (1) from lessened blood supply, (2) from deficient trophic impulse.

Surgically, atrophy from interference with the nerve supply is seen in cases of injury to or disease of nerves.

Effects.—In cases of simple atrophy the part retains its normal shape, but is smaller and lighter than normal; but as fatty degeneration is usually associated, these changes may be masked, and the part be much increased in size and weight. When the component cells of an organ atrophy, it may become much denser than natural, from an undue preponderance of connective tissue. If the walls of a hollow viscus atrophy it will dilate or even rupture. The functional activity of an atrophied structure is necessarily diminished, according to the degree of the change.

Treatment.—The removal of the cause and the promotion of healthy nutrition must be effected. Atrophied muscles should be electrically stimulated, their use encouraged, and nutrition favoured by cold affusion and massage. In unreduced dislocations it is important to bear in mind, during attempts at reduction, that the bone is atrophied and weaker than natural.

Hypertrophy

Hypertrophy implies that a tissue is simply overgrown; when the component elements are also increased in number, the condition is sometimes spoken of as Hyperplasia or Numerical Hypertrophy. The mere fact of enlargement of an organ does not necessarily imply hypertrophy, for in most cases the enlargement is dependent upon some other tissue, thus in so-called hypertrophy of the breast the greater bulk of the organ is formed of fat (False Hypertrophy).

Causes.—Hypertrophy usually occurs in response to some increased need for growth; thus the bladder wall hypertrophies in response to additional work thrown upon it by stricture of the urethra, or if one kidney be rendered practically functionless, the other increases in size to meet the demands upon it; in such cases the hypertrophy is said to be compensatory.

Intermittent pressure causes hypertrophy by inducing a periodic afflux of blood to the part, as is commonly seen in the case of corns, or thickening of the epidermis.

Occasionally some part of the body, especially the fingers or toes, is congenitally much larger than normal, from causes of which we are ignorant.

CHAPTER II

Local Circulatory Disturbances—Inflammation

Ischæmia

By ischæmia is meant a diminution of the blood supply of a part. This condition is sometimes clinically spoken of as anæmia, a term more fittingly reserved for cases in which the blood supply is absolutely cut off, or to indicate a general state due to poverty of the blood itself.

Causes.—Ischæmia is due to arterial obstruction, which may be produced by thrombosis, embolism, obliterative arteritis, ligature, or pressure from without. Ischæmia of nervous origin plays an important part in certain pathological conditions, of which Raynaud's disease is a notable example (see p. 82). Ischæmia is usually a temporary state, since the interference with the circulation through an artery is speedily compensated for by collateral circulation. Were it not for this adaptability of the vessels, surgical operations involving the ligature of large and important vessels would be impossible.

Chronic ischæmia is common in advancing life when calcareous and atheromatous changes in the vessel walls are pronounced, for not only are the vessels narrowed and the walls less elastic than normal, but in such cases thrombosis frequently supervenes.

Effects.—Diminution of the blood-stream limits the supply of nutritive material, and consequently lowers vitality.

Deficient nutrition naturally increases the proneness of the tissues to inflammation and other pathological lesions of a trophic nature; an ischæmic area is consequently ready to respond to irritation of slight intensity, which would produce no effect on healthy tissues; wounds heal with difficulty and degeneration soon occurs.

Anæmia

The term anæmia is here used to indicate complete cutting off of the blood supply in consequence of obstruction of the vessels. The causes of the condition are the same as those of ischæmia, but since, in the majority of cases, collateral circulation is speedily established, anæmia rarely occurs. Surgically, complete anæmia is seen in cases of strangulation of the bowel.

Unless quickly relieved, anæmia inevitably leads to gangrene.

Active Arterial Hyperæmia

Active hyperæmia is dependent upon relaxation of the muscular coat of the arteries, which may be induced (1) by direct stimulation causing a temporary paralysis from injury; (2) by the influence of the central nervous system excited by central changes or reflex action; (3) by injury or pathological destruction of the sympathetic nerve filaments.

Effects.—Active hyperæmia causes redness of the part, increased rapidity of the blood stream, with some elevation of the local temperature, and a sense of throbbing and fulness.

Active hyperæmia may be beneficial, as in the healing of wounds and the absorption of inflammatory products, but it may also pass into the inflammatory state.

Passive Hyperæmia—Venous Congestion

Causes.—Venous congestion may be due (1) to a defect in the propulsive forces of the circulation, (2) to obstruction in the veins.

The blood is driven onward by the action of the heart and the elastic recoil of the arterial walls, and hence any disease impairing the strength of these forces entails venous congestion proportional to the diminution of power. Mere feebleness of the heart's action, independently of any disease of its structure, may cause dangerous hypostatic congestion, as is frequently seen in those enfeebled by disease or old age.

Venous obstruction may be due to thrombosis, varicosity of the vessels, or pressure from without. Valveless and unsupported veins, especially if liable to periodic engorgement, are very prone to be the seat of congestion, *e.g.* the hæmorrhoidal plexus.

Effects.—Venous engorgement, if long continued, leads to very important results, and is one of the most potent predisposing

causes of inflammation. The part is of a dusky livid colour, and may be pigmented from the disintegration of escaped red blood cells. The temperature is diminished, and there is a sensation of coldness, numbness, and weight. Œdema varies considerably in amount, according to the extent and duration of the congestion and the power of the lymphatics to absorb the exuded fluid. The fluid bathing the tissues is poor in albumen and fibrin, contrasting with that poured out as the result of inflammation. Chronic congestion leads to induration from the formation of new fibrous tissue, which, by the pressure it exercises, still further impedes the circulation. It also induces chronic inflammation by lowering the vitality of the tissues, since it hinders the free interchange of oxygen and carbon dioxide.

Chronic eczema of the skin and ulceration are common results, and unless the congestion can be overcome, prove most intractable affections.

Congested veins may rupture, but rarely bleed much, partly because it is only the small vessels which give way, and partly because the feebleness of the circulation favours coagulation and spontaneous arrest.

Treatment.—The line of treatment must depend upon the actual cause of the congestion. If the force of the circulation is deficient, the heart's action must be encouraged by cardiac stimulants and such means as the special features of the case may suggest.

Pressure on the veins must if possible be removed. The circulation should be encouraged by the elevated position, cold douching, friction and massage, and the parts must be kept warm. Local bleeding is useful in some cases. The use of diuretics, saline purgatives, and diaphoretics serves to unload the venous system.

Inflammation

If there is one subject in General Pathology of greater importance than another, it is surely that of the Inflammatory process. Travers has justly said that "a knowledge of the phenomena of inflammation, the laws by which it is governed, and the relations which its several processes bear to each other, is the keystone to medical and surgical science." Its common occurrence, numerous causes, various phases and results, combine to render it a subject of extreme interest, and one with which the surgeon must be thoroughly familiar if he is desirous of understanding the greater part of practical medicine and surgery.

Inflammation has been defined by Burdon-Saunderson as being "the succession of changes which occurs in living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality." While this definition leaves much to be desired, it has the advantage of conveying a general concept of what we mean by inflammation without committing us to the adoption of any pathological dogma. The series of events, the sum of which constitutes the inflammatory process, have been repeatedly followed in the frog's web and tongue, the rabbit's ear, and the mesentery of both. These changes are similar in cold- and warm-blooded animals, but in the latter greater care is needful in the conduct of the necessary experiments.

Physiological data.—Under normal conditions the calibre of the arteries may be dilated or contracted under the influence of the vaso-motor nerves, either as the result of central or reflex irritation. Whether any peripheral vaso-motor nervous mechanisms exist or not is at present uncertain. Contraction of a vessel is due to active contraction of the circular muscular fibres, but dilatation is purely passive. Pathological changes, which impair the nutrition of the arterial coats, affect their elasticity and favour or produce dilatation.

The endothelial cells forming the walls of the capillaries are contractile, and hence the calibre of these vessels is capable of alteration. The cement substance between the capillary endothelial cells, when stained with silver nitrate, exhibits specks and slits called stomata, but whether or not these are really holes in the vessel wall is uncertain.

The blood in the minute vessels is divided into two zones—the axial and the plasmatic; in the small capillaries the differentiation between axial and plasmatic zones is not so evident as it is in the small arteries and veins. The axial zone consists of red blood cells, while most, but not all, of the white cells are found in the plasmatic zone, the rate of flow of which is much less than that of the axial current. The white corpuscles exhibit amœboid movements, and roll lazily along in close contact with the vessel wall. Under normal conditions the red blood cells remain discrete, but when their vitality is lowered they adhere to one another and thus offer increased resistance to the blood flow.

The fluid parts of the blood are constantly filtering through the capillary and venous walls, irrigating the tissues and being absorbed again by the lymphatics. A few leucocytes also escape and wander outside the vessels. No exudation takes place from the arteries.

Etiology.—Predisposing causes to disease are those conditions which, by impairing the vitality and hence the resisting power of the tissues, lay them open to morbid processes under slight provocation which more favourable and healthy conditions would enable them to withstand. The conditions essential to the maintenance of healthy nutrition have already been alluded to in considering the degenerations (p. 2), and further special causes of circulatory disturbance have been mentioned in connection with ischæmia and congestion (pp. 9, 10). The predisposing causes of inflammation must be sought in some purely local state, or in a general constitutional predisposition, *e.g.* the Strumous Diathesis. Chief among these causes are to be noted:—

(1) A deficiency in the supply of arterial blood.

(2) Venous congestion, and consequent stagnation of the blood-stream and failure of removal of the waste products of metabolism.

(3) Impurity of the blood or deficiency in its quality, either from accumulation of waste material (*e.g.* renal disease), the admixture of poisons (*e.g.* alcohol), or insufficient and improper food so frequently associated with bad hygienic surroundings.

(4) Interference with the normal nervous supply and trophic influences, *e.g.* acute bed-sores and perforating ulcer.

The general predisposing causes may be due, as in the case of the gouty or strumous diathesis, to some congenital and often hereditary vice of the tissues, or to acquired conditions such as may be induced by the abuse of alcohol or by unhealthy occupations or surroundings.

Exciting causes.—Anything capable of inducing inflammation is spoken of as an "irritant," but it is necessary to this end that such irritant shall act with a certain intensity, and for a certain time; these depending upon the presence or absence of predisposing causes. If the intensity of the irritant be great, it may kill the part to which it is applied, but the dead part will be surrounded by a zone of inflammation occurring in the tissues near enough to feel the effects of the irritant, but sufficiently remote to remain alive. This is well seen in the production of an eschar by the actual cautery.

Irritants of slight intensity may, when the tissues are unhealthy, *i.e.* predisposed, excite widespread inflammation, sometimes fraught with dangerous consequences. Every surgeon is familiar with the fact that even simple operations may have disastrous results in

drunkards, diabetics, or in those suffering from renal disease and some other general states.

Mechanical causes, such as wounds, friction, and tension, are common factors in inducing inflammation, and some of these at least are capable of being guarded against by the surgeon. The avoidance and relief of tension is of the utmost importance in surgical practice, with a view to preventing or arresting inflammation. If the tension of the exudate in an inflamed part is great and is not relieved, it tends to aggravate the condition, besides causing intense suffering. Some mechanical irritants arise within the body itself (*e.g.* calculi or sequestra), or may have been embedded in the tissues for a long time without having caused any harm (*e.g.* bullets, needles, splinters); in these cases the inflammation is conservative in its object, the result being the extrusion of the foreign body. It is generally believed that in such cases the resulting inflammation is dependent upon the presence of micro-organisms, which have gained entrance to the seat of the foreign body.

Heat and cold beyond certain limits (within which they exercise a beneficial effect upon the tissues) may excite inflammation by inducing vascular disturbance. Any chemical substance having irritating properties, such as the strong mineral acids, caustic alkalies, and certain vegetable substances, will excite a definite degree of inflammation proportional to the extent of their application. All the above causes are, however, of comparative insignificance as compared with the irritating effects of micro-organisms and their toxines, for a full account of which the reader is referred to the chapter on Bacteriology (p. 87).

Varieties.—Inflammation is classified according to its cause, extent, duration, and the essential feature of the phenomenon.

1. **Cause.**—Inflammations may be traumatic, gouty, rheumatic, tubercular, syphilitic, etc., according to the constitutional condition underlying the process. When no definite cause can be assigned, the process is sometimes said to be idiopathic. The distinction between simple, septic, and infective inflammation is of the greatest importance.

By *simple inflammation* we mean that which is not dependent upon any essential poison, and which is consequently always limited and never severe.

A *septic inflammation* is one dependent upon the irritation produced by the chemical products of decomposition brought about by micro-organisms; the process is proportional to the dose of the irritant, and ceases with its removal (p. 102).

Infective inflammations are due to the presence of pathogenic organisms, and may be locally or generally infective, according to their manner of spreading in the tissues (p. 103).

2. **Extent.**—*Local* inflammation is dependent upon some simple cause which acts temporarily on healthy tissues.

Spreading inflammation is due to invasion of the tissues or blood-stream by micro-organisms. A spreading inflammation may be excited in one of the following ways:—

(1) The tissues may be soaked with the irritating products of decomposition, the organisms not, however, spreading in them.

(2) Organisms of an infective nature may invade the tissues by continuity, as in the case of phagedæna.

(3) Organisms may locally invade the tissues and also spread by the lymph paths to the neighbouring lymphatic glands, *e.g.* cellulitis.

(4) The true general infective inflammation is due to infective organisms which may not only spread locally, but which, by finding their way into the blood-stream, either directly or through the lymph channels, become capable of exciting secondary inflammatory centres at distant parts.

For further information, the reader is referred to the chapter on the Infective Processes (p. 102).

3. **Duration.**—Inflammation may be acute, subacute, or chronic, according to the nature of the cause, the duration of its action, and the natural resistance or proneness of the affected tissues.

4. **Nature of the phenomena.**—Inflammation may be adhesive, *i.e.* the poured-out lymph organises and new tissue results, as in the union of wounds; or it may be suppurative, ulcerative, or gangrenous. The term phlegmonous implies that the local condition is very acute, the course being rapid and often ending in sloughing. Catarrhal inflammation attacks mucous surfaces and is described at p. 36.

The terms sthenic and asthenic are referred to at p. 31, footnote.

ACUTE INFLAMMATION

The phenomena of inflammation.—On the application of an irritant, the vessels in the affected area dilate. In some cases dilatation is preceded by initial contraction, which is quite transitory in its nature and of no importance as regards the ensuing changes.

Dilatation does not affect arteries, veins, and capillaries in an equal degree, the arteries showing the greatest enlargement and the capillaries the least. In consequence of this dilatation the general vascularity of the part is considerably augmented, and numerous small vessels which, on account of their minuteness, were before unnoticed, spring into prominence.

Coincident with this general dilatation there is increased velocity of the blood-stream, soon followed, however, by marked retardation

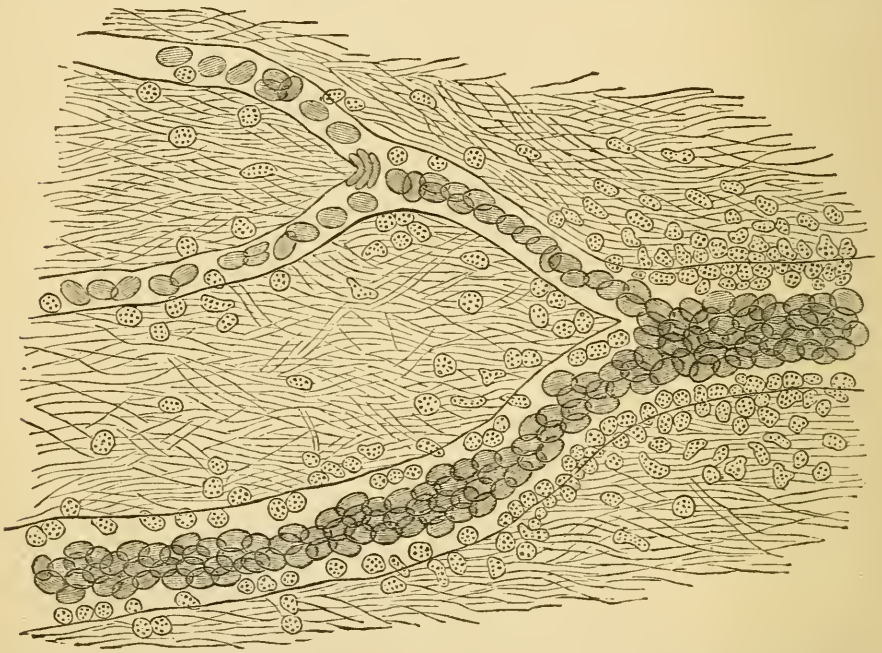

FIG. 3.—Veins and capillaries from the mesentery of a frog, after exposure for several hours. The axial stream of red blood cells is in circulation; the leucocytes have collected along the vessel walls and are wandering into the connective tissue of the mesentery (diapedesis). (Billroth's *Surgery*.)

culminating in complete stasis, which is preceded by oscillation as the velocity of the flow is temporarily increased during the cardiac systole. If the inflammation is severe and lasts long enough, stasis is followed by thrombosis.

The leucocytes, separated from the axial stream, crowd the plasmatic layer, line the vessel wall, and move slowly along during systole. This disposition of the white cells and their escape from the vessel, as below described, is chiefly seen in the venules (Fig. 3); in the small arteries and capillaries they are not so numerous, and do not form such a continuous layer in the plasmatic zone; moreover, they are mixed with red cells, which predominate in number.

The leucocytes escape through the walls of the veins and capillaries (diapedesis). If a small vein is closely watched, it will be seen that minute projections are visible on its outer surface, and that these correspond to leucocytes in the interior; in the course of time these irregularities of outline become more evident, the leucocyte being proportionately less so. In point of fact, the leucocyte is actually passing through the wall, to which, having

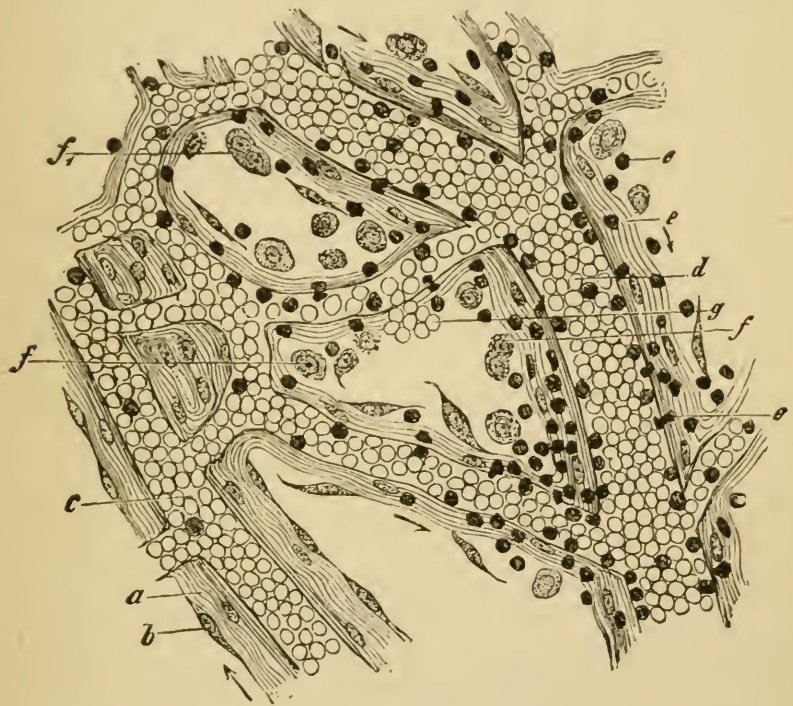

FIG. 4.—Inflamed omentum (Ziegler). *a*, fibrous tissue; *b*, epithelium; *c*, artery; *a*, vein with leucocytes along its wall; *e*, migrating and migrated leucocytes; *f*, desquamating and degenerating epithelium; f_1, proliferating epithelium; *g*, escaped red blood cells.

escaped, it is at first attached by a minute process, but soon wanders freely into the surrounding tissues. Numberless leucocytes thus escape and infiltrate the tissues. Red corpuscles also escape in small numbers in consequence of the intravascular pressure, but being non-motile, they remain in close proximity to the vessel from which they have passed unless carried to distant parts by the fluid exudate (Fig. 4, *g*). Diapedesis is confined to the veins and capillaries; it does not occur from the arteries, and ceases with stasis. Coincident with diapedesis there is an escape of fluid exudate which bathes the tissues. This fluid exudate, mixed with the leucocytes,

is known as inflammatory lymph. It differs from that which normally bathes the tissues, in that it is richer in albumen; it also rapidly and firmly coagulates in the tissues, the serum, as it separates from the coagulum, being taken up by the lymphatics. The precise nature of the inflammatory exudate is not always the same, its composition varying with circumstances concerning which we are not as yet fully informed. The more vigorous the health of the individual, the more fibrinous and readily coagulable is the exudate, whereas in the weak and enfeebled the fibrin is deficient in quantity.

The seat of the inflammation also modifies the exudate; thus in inflammation of mucous membranes, sero-purulent exudation is met with; while in that of serous membranes, firmly coagulable fibrinous exudation is common. The cause of the inflammation has also some influence in determining the nature of the exudate. Sometimes the effusion is chiefly serous and, although containing fibrin, does not show any tendency to coagulate; such is the case in rheumatic synovitis or hydrocele. Such a condition is favourable, since coagulation of the inflammatory product prevents its ready absorption; fluidity, *per contra*, favours, or at least does not hinder it. The ordinary sero-fibrinous exudate varies in the relative amount of fibrin and cells. All inflammatory effusions may be mixed with more or less blood, especially if the inflammation occurs as the result of injury, or in those of debilitated health, and notably in scurvy and hæmophilia. Inflammatory effusion may be mixed with the normal secretion, if any, of the part inflamed; thus in the case of mucous membranes, mucus is present, or in that of a joint, synovium.

Effect upon the tissues.—The effect upon the fixed tissue-cells of the inflamed part varies with circumstances, but in all cases the inflammatory exudate causes pressure upon them, and this, coupled with the vascular stasis, necessarily entails loss of vitality which may, unless the process be arrested, lead to destruction. Death of the tissues may also be due to the strength of the irritant, or to its direct corrosive action. The fixed cells do not multiply during the height of the inflammatory process; but when this has subsided, and repair or resolution sets in, they do so and give birth to phagocytes and new tissue elements.

Short of death, the cells may, from nutritive disturbances, undergo degenerative changes, softening, and absorption, their place being taken by the inflammatory exudate.

Behaviour and fate of the exudate.—The albuminous fluid

exudate which infiltrates the tissues in an inflamed area speedily forms a dense coagulum, clotting being favoured by the altered vitality of the tissues and by the presence of the numberless leucocytes, many of which are broken down, and hence free the fibrin-ferment. The further away the exudate gets from the point of irritation the less is the liability to coagulation, and the fluid is absorbed by the lymphatics. When clotting occurs, the fibrine and leucocytes entangled in its meshes form dense inflammatory lymph which, on removal of the irritant and the consequent arrest of the inflammation, gradually becomes absorbed.

According to Metchnikoff and the phagocytic theory, the

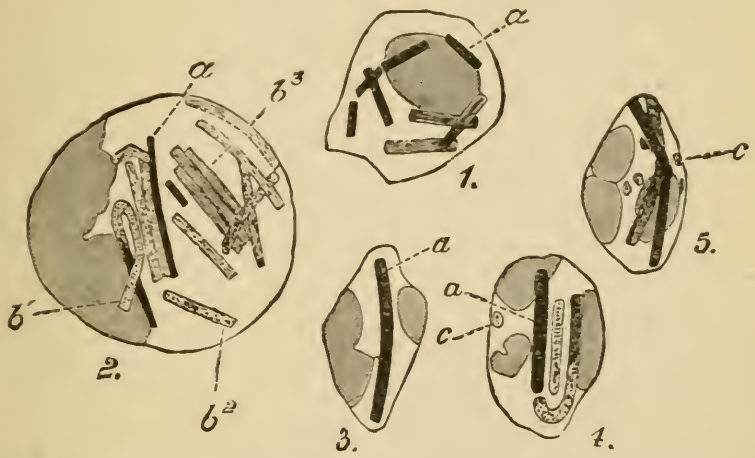

FIG. 5.—Anthrax of pigeon (an animal only slightly susceptible to the disease) to show the stages of destruction of bacilli by phagocytes. 1, macrophage from exudation from the eye of refractory bird; 2, macrophage from muscle of region of inoculation of bird that succumbed; 3, 4, 5, microphages from the eye twenty-seven hours after inoculation; *a*, *a*, unaltered bacilli; b^1, b^2, b^3, bacilli becoming more and more degenerated and indistinct; *c*, *c*, débris of bacilli. (Allbutt's *System of Medicine*, after Metchnikoff.)

escaped leucocytes crowd round the noxious agents (usually micro-organisms) and engage in a war of supremacy with them, and also remove, by ingestion, any dead cells or other matter (*e.g.* blood clot) which may be present (Fig. 5). While all pathologists agree that the leucocytes can perform this latter office of scavengers, many dispute their power to remove living organisms, or at any rate deny that this action is the *raison d'être* of their crowding into the inflamed area.

As regards the ultimate fate of the leucocytes, Metchnikoff asserts that, having accomplished their purpose and destroyed the micro-organisms, they themselves fall a prey to large phagocytes born of the fixed cells of the part and the endothelial cells of the

lymphatics. Many of the leucocytes die and become disintegrated during this war for supremacy, their remains serving as pabulum for the still living phagocytes.

Explanation of the phenomena—Pathology.—At the present time pathologists are divided as to the precise interpretation to be put upon the facts observable in inflamed areas.

While one school, following the teaching of Cohnheim, lays the responsibility primarily on changes in the vessel walls, Metchnikoff and his disciples assert that inflammation is due to phagocytic reaction on the part of the leucocytes. To use Metchnikoff's own words: "The *primum movens* of inflammation consists in a phagocytic reaction on the part of the animal organism. All the other phenomena are accessory to this process, and may be regarded as means to facilitate the access of phagocytes to the injured part." The blood-vessels, to damage of which Cohnheim attributed the inflammatory process, are, according to Metchnikoff, not necessary for its production, although the vascular disturbance materially assists by bringing phagocytes [1] to the injured area. Ziegler, opposing Metchnikoff's views, says that "The phagocytosis which occurs in the course of an inflammation is a purely accidental phenomenon, which is brought about for the simple reason that motile cells happen to be present, together with a material capable of being ingested by them." The theory of phagocytosis and its relation to the inflammatory process will be fully discussed later (p. 97).

Inflammation must doubtless be regarded as a physiological rather than a pathological process; it is the sum of those changes which are consequent on irritation, and which have for their object the removal of the offending *materies morbi;* this being accomplished, all the phenomena of inflammation disappear.

The initial contraction of the blood-vessels, which may occur immediately on receipt of the injury inducing the inflammation, arises from direct stimulation of the muscular coat; the subsequent dilatation is purely passive, and is due to paralysis from injury. While paralysis from direct injury to the vessel wall is the chief cause of the vascular dilatation at the point of action of the irritant, reflex nervous irritation or local nervous mechanisms play a part in causing the vascular dilatation, especially in those vessels remote from the seat of irritation. The increased velocity of the blood-stream in the inflamed area is due to the fact that all the vessels are not equally dilated. The amount of blood brought to the part

[1] Phagocytes are cells derived from the leucocytes and endothelium of the vessels and lymphatics (see p. 98).

by the widely dilated arteries must flow more quickly in order to pass through the capillaries, which show but little alteration in size.

Retardation of the stream and subsequent stasis cannot be due to diminution of the propelling force, since no such diminution is present; indeed, the force of the heart-beat is usually increased. The vessels being dilated, the size of their lumen cannot give rise to increased local resistance, and yet there is such an increase. In accordance with the supposition that all the changes in inflammation are due to changes in the vessel walls, retardation and stasis are looked upon as consequences of some chemico-physiological change in the vessel walls, whereby the accumulation of leucocytes and massing of red cells into rouleaux is favoured, thus increasing friction and offering a mechanical impediment to the blood flow.

Diapedesis is due to the inherent amœboid activity of the leucocytes, aided, perhaps, by increased intravascular pressure. According to Cohnheim, the escape of leucocytes and lymph is dependent upon the altered state of the vessel wall, whereas Metchnikoff regards it as the essential feature of the inflammatory process, and dependent upon an attractive influence (positive chemiotaxis) exercised on the cells. In support of his contention, the latter authority points out that diapedesis does not always occur. Thus he instances tuberculosis: If an animal be inoculated with the bacillus under the skin, inflammation with diapedesis and the formation of extravascular tubercle occurs; but if the inoculation be intravascular, the leucocytes gather round the bacilli within the vessel and intravascular tubercles result. In fact, the leucocytes collect wherever the noxious irritant is present.

The fluid exudate, rich in albumen, escapes in consequence of the increased vascular tension and the alteration in the vessel walls; under normal conditions, albumen does not pass through the vessel walls, which exert a selective influence on the filtrate, but during inflammation the vitality of the tissues is lowered and consequently no bar is offered to the escape of albumen.

Termination of acute inflammation.—As the inflammatory process is merely the succession of changes occurring in the tissues in response to some form of irritation, its duration, extent, and method of termination necessarily vary with the nature of such irritant. As soon as the exciting cause has been removed, the inflammatory process is arrested; in mild cases this happens before any appreciable damage has been inflicted, and the part is completely restored (Resolution); in more severe cases some destruction of tissue occurs and repair must follow (Granulation, Organisation);

in the severest forms this destruction is accompanied by the formation of pus (Suppuration, Ulceration), and perhaps appreciable portions of tissue, being completely deprived of blood, die *en masse* (Sloughing and Gangrene).

It must not be forgotten that the essential nature of the cause is not the only factor, although by far the most important one, to be reckoned with in forming an opinion as to the progress of the inflammation, for a good deal depends upon the state of the tissues themselves. In weakly tissues an irritant of slight intensity may occasion considerable inflammatory action and consequent damage, whereas the same irritant would not appreciably affect healthy tissues, or at most would only induce a mild form of inflammation speedily terminating in resolution.

As an example we may instance the special liability to destructive inflammation in congested parts and in patients suffering from diabetes, and the well-known facts of predisposition of individuals to certain diseases due to micro-organisms, *e.g.* erysipelas.

Resolution.—When the inflammatory process has been of mild intensity and the irritant inducing it has been removed, the vitality of the tissues and vessels is restored and a *status quo ante* is brought about. The nutritive equilibrium of the vessel walls is regained, and hence they resume their normal tone and power of contraction and dilatation under the vaso-motor mechanism. The circulation is re-established, the corpuscles nearest the still-flowing blood gradually breaking away from the stagnant blood until the stream in the previously inflamed area returns to the normal.

The vessel walls regain their filtering action and no longer permit the free escape of lymph and leucocytes, whatever exudation is allowed being kept within normal bounds. As regards the inflammatory exudate, the fluid part and many of the white cells are quickly absorbed by the lymphatics; any red cells which may have escaped disintegrate and are removed by phagocytes; the coagulated fibrin and such cells as are entangled in its meshes are removed by the fixed phagocytes derived from the endothelial cells. Sometimes the amount of exudation is so great that its removal by absorption is very slow or incomplete; in such cases surgery may assist, as in pleural effusion or synovitis, by drawing off the superabundant fluid. If the coagulated exudate is very large in quantity, the area of inflammation may remain indurated for a long time, or the exudate may undergo fatty changes leading to caseation. This process will be more fully mentioned in dealing with chronic inflammation.

Granulation and organisation. — When inflammation has caused destruction of tissue by absorption, or as the result of suppuration, and when the process itself has been arrested, repair of the damaged tissue sets in.

Organisation may be a beneficial or harmful process, according to the situation in which it occurs; beneficial, if it repairs a loss of tissue, as in the healing of a wound or abscess; harmful, if the new tissue replaces one of higher physiological rank, whose functions the new connective tissue is incapable of fulfilling.

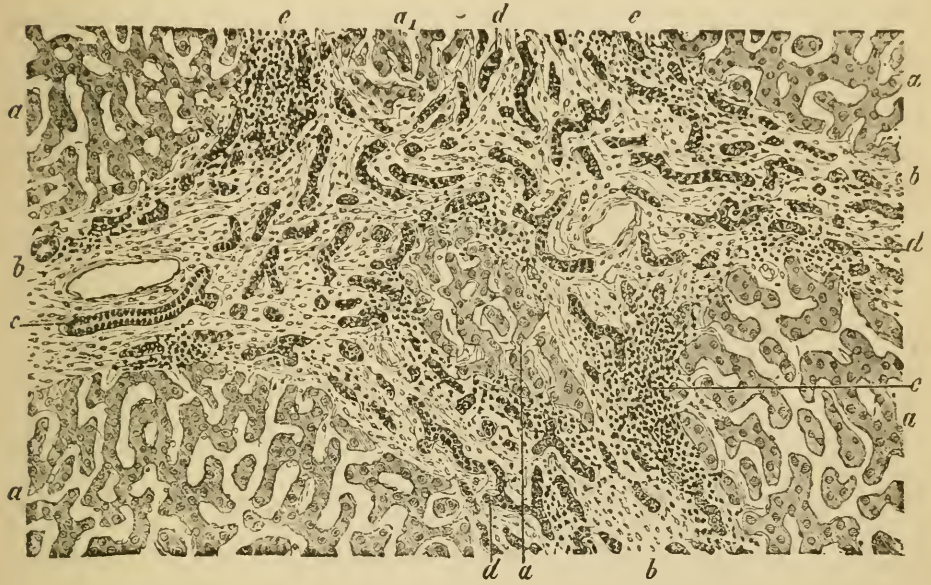

FIG. 6.—Formation of scar tissue and new bile ducts in chronic hepatitis (Ziegler). *a*, a_1, hepatic lobules; *b*, new scar tissue; *c*, old bile ducts; *d*, newly-formed bile ducts; *e*, round-celled infiltration.

Repair is said to be perfect when the new tissue is similar in its chemical, physical, and physiological properties to that which it replaces. Man's power of perfect repair is far inferior to that of many of the lower animals; thus, lizards will completely regenerate their tails and limbs, and some worms have yet more perfect powers of regeneration.

When repair is imperfect, the loss to the economy varies within the widest limits; thus, if a portion of the breast substance has been destroyed and replaced by connective tissue, the harm done is practically not felt, since the breast cannot be regarded as a very important organ; but if a similar condition were to affect the conducting paths of the spinal cord or any other organ of vital im-

portance, the resulting loss would be disastrous and widespread; between these two instances many gradations of mischief will readily occur to the mind (see p. 34).

For an account of the process of repair, the reader is referred to chap. ii. vol. ii.

Suppuration—Destructive inflammation.—If the irritant is of sufficient intensity to cause necrosis of any cells, the part speedily becomes infiltrated with leucocytes. The dead area softens, the cells disintegrate, and a minute abscess is formed which gradually increases in size. The process is fully described at p. 38.

The occurrence of suppuration is in the main to be regarded as unfavourable; in some cases, however, it is beneficial to the patient, as being one of Nature's methods of freeing the body from such injurious irritants as a foreign body or sequestrum.

Local signs of acute inflammation.—The association of redness, swelling, heat, and pain with, in many cases, functional derangement, is characteristic of inflammation; one or more of these signs may be met with under other conditions, but all are present in the inflammatory process, although they do not necessarily manifest themselves in equal degree.

Redness is dependent upon vascular engorgement, and hence is most marked in parts of high vascularity, but is not visible in the case of internal inflammation. The intensity of the colour and its distribution vary. In the skin the whole area is the seat of the blush, but in mucous membranes the individual vessels may show up as bright streaks and the surface is said to be injected. In very intense inflammation, some of the redness is dependent upon the escape of red cells and capillary hæmorrhage. If the inflammation has reached that stage in which the stagnant blood is thrombosed, pressure will not diminish the colour; in other cases the blood is driven onwards, but the blush returns as soon as the pressure is relieved. The margin of the redness gradually fades into the normal colour of the part, except in certain cases (*e.g.* cutaneous erysipelas) in which it is sharply outlined. With subsidence of the inflammation the redness disappears, but some pigmentation may remain if red cells have escaped from the vessels.

Swelling is due to the inflammatory exudate and, in a slight degree, to the vascular engorgement. In health, the normal exudation from the vessels is taken up by the lymphatics, and during inflammation this absorption is increased; but as the exudate coagulates in the tissues, its complete removal by the lymphatics becomes impossible. The amount of exudate is dependent on

the intensity of the inflammation and the natural vascularity of the part. In lax vascular tissues, *e.g.* the scrotum or face, the swelling is often enormous, since there is a large quantity of exudate and but little tension. In dense fibrous structures such as tendon-sheaths, the exudate is at high tension, and hence the swelling is not marked. The exudation naturally travels in the direction of least resistance, permeating the connective tissue of the part, and, in the case of cavities, being poured into the sac; in the lungs it fills the alveoli, and, on serous membranes, forms a tough layer of lymph with serous accumulation. The degree of swelling in all these cases is necessarily subject to great variation, and is sometimes practically inappreciable.

Heat.—The local heat is due to the increased afflux of arterial blood. The degree of heat, as complained of by the patient, is merely a measure of sensation, and is greater than that indicated by the surface thermometer, for although there is certainly an appreciable rise in the local temperature, yet this never reaches the temperature in the rectum.

Pain.—Pain is due to pressure of the inflammatory exudate, and, in open wounds, to exposure of the nervous filaments or to their irritation by the chemical products of decomposition; in some cases neuritis adds much to the suffering. Individual susceptibility also plays an important part. As pressure is the main cause of the pain, the latter will, other things being equal, be dependent upon the amount of the exudate, but it is also very materially influenced by the natural nerve supply of the part. In lax tissues, where the exudate can easily distend the parts without causing much tension, pain may be very slight in spite of great swelling and a plentiful nerve supply.

On the other hand, the pain caused by inflammation of dense structures is severe, as is seen in the case of whitlow, periostitis, or orchitis.

The pain of inflammation is usually dull, aching, and throbbing in character; it is increased by the dependent position which favours engorgement of the inflamed part, and is worse at night, probably because the nervous system is more impressionable. Very often the pain has a "tensive" character, conveying to the patient the idea that something is pent up to which exit should be given.

Alteration in function is due (1) to the altered vital condition of the inflamed tissues, (2) to the mechanical impediment caused by the accumulated exudate, and (3) to the pain caused in parts con-

cerned with movement. The function of the part is generally diminished or lost for the time being; in other cases it is perverted. Thus, the bladder has two distinct functions—it serves to hold urine and also to expel it; but when its walls are inflamed, the viscus can only hold a very small amount of urine, while its expulsive efforts are frequent and strong. Similarly, in the case of the special senses, the function is not only diminished but perverted, so that there are auditory, visual, or other hallucinations according to the sense affected.

The constitutional symptoms of acute inflammation.—Inflammation of slight intensity, especially if attacking an unimportant part, is not accompanied by general disturbance; but when a certain degree of severity is reached, certain symptoms make themselves manifest, the sum of which constitutes the febrile state. The degree of fever and the gravity of the general disturbance vary with circumstances; the physiological importance of the part, the intensity and extent of the mischief and, above all, its cause being the most important factors. The young and the aged, and those of a nervous temperament, are liable to high fever from slight causes.

As regards situation, inflammation of the lungs, brain, kidneys, tonsils, and ear is accompanied by severe constitutional symptoms; whereas a similar affection of a joint or bone occasions but little disturbance. The precise nature of the process is of the greatest importance, a simple inflammation producing much less serious effects, even when widespread, than does one which is dependent on some infective organism, or is associated with putrefaction and retention of the discharges, for in such cases the absorption of toxines produces more or less severe general disturbance.

The physiology of heat.—In warm-blooded animals the production and loss of heat are so regulated that a fairly uniform temperature is maintained. Heat is produced by oxidation and combustion, the greater quantity being formed in the muscles, abdominal viscera, and brain, *i.e.* in parts exhibiting great physiological activity.

About 75 per cent of the heat is lost by evaporation and radiation from the skin, 18 per cent by the lungs, and the remaining 7 per cent by warming the excreta.

The maintenance of a normal temperature is dependent upon a balance being struck between production and loss, which is brought about by the influence of the nervous system. Experiments and clinical observation tend to show that there is a thermogenic centre

in the brain, probably in the pons. The production of heat must also be dependent upon trophic influences, themselves of nervous origin; its loss may be increased or diminished by alterations in the breathing, or in the vascular engorgement of the skin.

The production of fever.—If the amount of heat produced is in excess of that which is lost, the patient becomes febrile; such a disproportion is dependent upon increased heat production, and not upon diminished loss. That production is increased is proved by the fact that although during the febrile state the patient takes less food, yet the discharge of urea and CO_2 and the rapid wasting of the body indicate increased oxidation; moreover, during the febrile state, the patient loses much more heat than under normal conditions.

How is this increased production of heat brought about? Undoubtedly through the intervention of the nervous system, the heat-producing centres being stimulated by certain substances circulating in the blood, and which, from their capability of inducing fever, are called "pyrogenic."

In simple inflammations, such as may occur in cases of fracture or subcutaneous injury, and in which there can be no question of septic or infective influences, the pyrogenic material is the fibrin-ferment which, as we have already seen, is contained in the serum, being set free by disintegration of some of the leucocytes. Fever dependent upon this cause is usually slight and transient, and if the exuded serum is very small in quantity, or is drained away, no fever results; under contrary conditions the temperature may remain high, rapidly falling, however, as soon as the serum is given free exit.

Fibrin-ferment is by no means the only pyrogenic material, nor is it a very important one. The products of putrefactive decomposition and of pathogenic organisms (Ptomaines and Toxines) are strongly pyrogenic, causing high fever with proportionate constitutional disturbance.

Fever due to the action of non-pathogenic or putrefaction fungi is directly proportional to the dose of the poison absorbed, and if this be drained away and the wound cleansed, the temperature quickly falls; but in the case of the pathogenic organisms such removal is often impossible, and so long as the organisms live and flourish in the tissues, so long are their toxines absorbed and excite fever.

Whether these poisons cause fever by acting on the thermogenic centres, or whether, circulating in the blood, they act directly on

the tissues independently of, or in association with, the nervous system, cannot at present be determined.

The symptoms associated with fever. — In any disease associated with fever, no matter what its precise nature or seat, there are, in addition to the symptoms which these latter may determine, certain constitutional effects due to the fever itself, such effects being as a rule proportional in severity to the height of the temperature. In cases where the temperature rises gradually, the symptoms develop gradually; but if the rise of temperature is rapid—as in many of the infective processes—the constitutional disturbance is equally sudden. Fever may be continuous, remittent, or intermittent, and the symptoms follow much the same course.

A sudden onset is usually ushered in by shivering or by a more or less severe rigor, followed by profuse sweating and rapid decline of the temperature, which may, however, quickly rise again. In young children a convulsive attack takes the place of a rigor. When the onset is gradual, the patient may complain of alternate shivering and flushing, with a sense of general malaise.

In moderate degrees of fever (103° F.) the skin is flushed, hot and dry, sometimes perspiring; the face is flushed and often anxious, the eyes are bright and suffused, and the alæ nasi may move slightly with respiration.

Alimentary tract.—The tongue is moist and coated, especially along the dorsum, with a creamy white fur. The mouth is clammy, and there is considerable thirst with loss of appetite, nausea, and perhaps vomiting. The bowels are constipated.

Urinary system.—The urine is scanty and concentrated. The specific gravity is increased, the colour heightened, and on cooling, the urine is rendered cloudy from precipitation of urates. The amount of urea and nitrogenous bodies is increased, but the chlorides are diminished.

Respiratory system.—The respiration is slightly increased in rapidity, but is shallow. Marked difficulty points to some affection of the lungs or pleura. The breath is often offensive.

Circulatory system.—The pulse rate is increased, usually about 5-10 beats for every degree of fever; increase beyond this ratio indicates cardiac failure. The pulse is bounding, soft, compressible, and often distinctly dicrotic.

Nervous system.—There is frontal headache, irritability of temper, wakefulness at night and occasionally slight delirium. Should delirium supervene, headache ceases, except in cases of

meningeal or cerebral mischief in which the patient complains of headache throughout.

When fever is continued for some days, or when it runs as high as 104° or 105° F., the symptoms are essentially the same, although some are modified. Nervous prostration is more marked, and delirium is usually present at night; the tongue becomes dry and cracked, and the lips and teeth are loaded with sordes, the patient being too feeble to close the mouth. The facial appearance indicates severe illness; the expression is anxious, the eyes dull and lustreless, and the complexion leaden.

In very severe cases, especially in those due to some acute infective process, the patient falls into the "typhoid" state, a condition of serious import.

Nervous prostration is extreme; the patient lies on his back, sunk down in the bed, the mouth is half-open, the eyelids drooping, and the face has a dull, leaden, expressionless aspect. The tongue is deeply coated with a dry brown fur, here and there deeply cracked, and sordes accumulate round the lips and gums.

There is profound mental apathy, and although the patient may usually be temporarily roused, he speedily relapses into indifference as to his surroundings. He takes fluid nourishment well.

This apathetic condition is accompanied by low muttering delirium (Typhomania).

Profuse sweating and diarrhœa are common, and add to the general prostration; the patient exhales a disagreeable earthy odour, and his hands have often a withered, shrunken appearance and brown discoloration.

As the end approaches, there is muscular tremulousness, subsultus, and risus sardonicus; the extreme nervous and muscular prostration is further evidenced by incontinence of fæces and urine. Finally coma supervenes and death closes the scene.

General prognosis of fever. — The prognosis necessarily depends upon the actual cause inducing fever, but at the same time the course the temperature runs may in itself be matter for anxiety.

In the case of continuous fever a sudden extra-elevation usually indicates the occurrence of some additional mischief or complication, *e.g.* suppuration; a sudden fall may mean convalescence, or else a complication inducing extreme collapse, *e.g.* perforation of the gut.

A temperature of 105° F. is in itself a serious matter; should hyperpyrexia (106° F.) set in, the case will almost certainly terminate fatally.

To be of any real value, at least two thermometric observations

must be taken daily (morning and night). In some diseases, *e.g.* tubercle, the degree of fever is so slight that the thermometer alone can detect it, its persistence indicates that the disease is in active progress, although the mischief may be so insidious that no other sign is certainly diagnostic.

The prognosis as to the duration of fever depends upon the circumstances of the case, and whether or not these are such as to permit of our getting rid of the supply of pyrogenic material; thus, in fever due to septic absorption, free drainage and flushing with an antiseptic lotion will speedily reduce the temperature.

Treatment of acute inflammation.— **Preventive.** — In surgical practice the preventive treatment of acute inflammation is a matter of paramount importance. It has already been stated that the most important and harmful class of irritants are the micro-organisms and their products, and hence the exclusion of these, or the removal of their products by the practice of antiseptics, is of the first importance (see chap. i. vol. ii.).

It is necessary, moreover, before performing any operation, to see that the general health of the patient and the vitality of the tissues to be operated on are good, otherwise, even an aseptic operation may lead to unfortunate consequences. When applying any apparatus, *e.g.* splints, great care must be taken not to cause injurious pressure or tension, both of which are exciting causes of inflammation.

Curative.—The tissues have a natural tendency to spontaneous cure, provided they are relieved from injurious influences; hence, if the cause of the inflammation can be removed, the process quickly subsides. This is unfortunately not always possible, and we have therefore to adopt local and constitutional remedies for promoting local and general vitality, and so aiding recovery. A knowledge of the causes of any pathological process and its phenomena is the basis of all rational treatment. It is very important to estimate correctly the relative degree of mischief dependent on local and on general causes; otherwise we may apply treatment in the wrong direction. Thus, constitutional treatment would be valueless in the case of an inflammation dependent on local irritation, and conversely, local treatment would do little good in the case of a syphilitic sore, unless constitutional remedies were also prescribed.

General means are directed towards the treatment of any dyscrasia present, or, in its absence, towards the maintenance of the patient's health, and the abatement of the constitutional symptoms during the acute inflammation.

Some forms of inflammation are successfully combated by certain drugs which are said to have a specific action, *e.g.* mercury and iodides in syphilis, salycine in rheumatism. Unfortunately the number of these specifics is very small.

The treatment of the constitutional effects of inflammation varies according to the strength and age of the patient and to whether the signs present are of the sthenic or asthenic type.[1]

In *sthenic cases* the bowels should be opened and kept acting by saline aperients with a view to promoting a free flow of fluid into the intestinal canal, thus diminishing blood pressure, and also to removing from the body effete material. As toxines are eliminated by the kidneys and the skin, the secretion of urine and sweat should be encouraged by the use of diuretics and diaphoretics; the citrate and nitrate of potash, Dover's powder, antimony and aconite are the most useful drugs for this purpose. All preparations of opium must be given with great care, especially if there is any renal mischief. If the inflammation is very acute, and the patient young and full-blooded, bleeding from the median basilic vein may do much good, notably in acute pneumonia and inflammatory conditions within the skull; but bleeding should not be practised at the two extremes of life, when the loss of blood is badly borne. Stimulants are rarely required.

In *asthenic cases* the main indications are to rid the system of any toxines which may be circulating in the blood, and to build up the patient's strength. Bleeding must never be resorted to; and although the action of the bowels, skin, and kidneys must be encouraged, drugs to attain this end must be cautiously administered, as their too free use further taxes the patient's failing strength.

Food must be given in an easily digestible and concentrated form—chicken and beef jelly, Brand's essence, Liebig, Carnrick's peptonoids, and Benger's food are of great use. Alcoholic stimulants are always necessary, and narcotics, especially opium, are indicated by the great nervous prostration. The pulse is the best guide as to the necessity for alcohol. If the heart is failing, alcohol must always be given in quantities sufficient to increase its strength. As a rule, nervous prostration and cardiac failure go together, and the effects of alcohol in inducing sleep and improving the nervous

[1] No very accurate definition of these terms can be given. A sthenic inflammation is one in which the local and constitutional signs assume an active form, and, although perhaps running a severe course, are strongly combated by the patient; asthenic inflammations are specially marked by intense nervous prostration and the rapid onset of "typhoid" symptoms, the patient showing little or no recuperative or resistant power.

tone are often remarkable. It should, as a rule, be held in reserve until clearly demanded by the state of the pulse, and when its object has been attained the quantity should be diminished.

By clinical experience, and by that alone, will the surgeon be able to adopt the happy mean between the depressant and stimulant line of treatment; and it is to the constitutional symptoms that we must look for guidance. A failing heart, nervous prostration, and adynamia, with symptoms merging into the "typhoid" state, are indications for free stimulation.

During convalescence the diet must be generous, and malt liquors may be given with advantage.

Local treatment.—*Rest* is one of the most important therapeutic agents we possess; it encourages repair and healthy nutrition, lessens vascular excitement, and favours absorption of the exudate. The method of obtaining rest necessarily varies with the seat of the mischief. Thus, in a joint we employ some form of splint; for the eye, atropine and avoidance of light. The principle remains the same in all cases, although the methods employed are as diverse as are the functions of different parts of the body.

Position.—When possible, the inflamed part should be raised, with the view of lessening congestion, and favouring the return of blood and lymph. Patients in many instances voluntarily raise and rest an inflamed part, since the pain is thereby much diminished.

Heat may be employed in the form of poultices, hot fomentations, hot baths, or by enveloping the part in cotton wadding. Whenever there is an open wound, hot antiseptic fomentations or baths should always be the method adopted.

Heat, especially combined with moisture, causes general relaxation of the tissues and dilatation of the cutaneous and other vessels, and hence relieves congestion of the inflamed area, favours absorption, diminishes tension, and relieves pain. It increases exudation from the vessels, and, if suppuration be imminent, hastens the process; if sloughs are present, their separation proceeds more rapidly, as heat increases the vitality of the tissues and so favours repair. Open septic wounds are rapidly cleansed by hot antiseptic irrigation with weak boracic solution or Condy's fluid. By this means the putrefying discharges are continuously washed away.

Cold may be applied by the ice-bag, Leiter's tubes, cold irrigation, evaporating lotions, or cold compresses. The ice-bag is usually the most convenient form. Leiter's tubes are very useful when cold has to be applied to the head or spine. Evaporating lotions are not much used, as they require constant reapplication.

Cold is especially useful in inflammation of the central nervous system, and in acute synovitis; it should be continuous, is only to be applied during the earlier stages of the inflammatory process, and never when suppuration is imminent. Cold diminishes congestion and exudation; but, generally speaking, is inferior to heat as a therapeutic agent.

Local sedatives are sometimes employed in conjunction with moist heat, but their beneficial effect has perhaps been over-estimated. Glycerine and extract of belladonna in equal parts, aconite, opium, or poppy-head fomentations are the usual remedies.

Local astringents are very useful in catarrhal inflammation of the mucous membranes (see p. 36).

The relief of local tension may be obtained by local bleeding, dry cupping, or by free incision aided by elevation, heat, and rest. Cupping is not much used in surgical practice. Leeches are useful in very acute inflammation with threatened suppuration. They should never be applied to any part where there is no resistant structure against which pressure may be employed to arrest the bleeding; this, if very troublesome, may be stopped by the application of a point of silver nitrate, or by acupressure. Bleeding by puncture of the veins is sometimes necessary in acute orchitis.

The most satisfactory method of bleeding to relieve tension is by incision, which allows free escape of the inflammatory exudate. This treatment is especially useful in periostitis, cellulitis, and spreading inflammations generally. When suppuration has occurred, a free incision into the abscess limits the destructive process.

CHRONIC INFLAMMATION

Etiology.—The causes of chronic inflammation are of the same nature as those inducing the acute form; but they act with less intensity and over a longer period of time, and whereas acute inflammation is much more dependent upon local causes, the chronic form is due in the main to general constitutional conditions, such as syphilis, gout, or struma.

The chief local condition favouring chronic inflammation is passive venous congestion, which lowers the vitality and resisting powers of the tissues by preventing the free interchange of oxygen and carbon dioxide, and hence favours the accumulation of waste and deleterious products.

In many cases chronic inflammation depends upon the association of several causes, each in itself insufficient to induce the change.

This is well exemplified in the case of chronic ulcers of the leg; venous congestion, enfeebled health, slight mechanical injury, such as a scratch, and too often neglect and uncleanliness, combining to produce a destruction of tissue which no one of the conditions would have caused *per se*.

The process and its results. — The series of changes characteristic of acute inflammation are also present in a modified form in the chronic process. The stagnant blood frequently coagulates, the engorged vessels rupture, and the blood is extravasated, the disintegrating red corpuscles causing subsequent pigmentation. The exudation is mainly cellular, the surrounding tissues being infiltrated with small round cells derived mainly from the leucocytes, but also from the fixed connective tissue cells.

The ultimate result depends in great measure upon whether the cause be simple, infective, local, or constitutional.

Complete resolution sometimes occurs; but more usually some induration, due to fibroid overgrowth, results. Organisation is common, the inflamed part being occupied by new scar tissue which, as it contracts, exerts injurious pressure on the proper cells of the part (Fig. 6, p. 23). The latter may in consequence undergo fatty degeneration, atrophy, and ultimate absorption, *e.g.* cirrhosis of the liver. The result of this change necessarily varies according to the seat of inflammation and the importance of the cells thus replaced by new connective tissue.

Organising chronic inflammation of the skin, subcutaneous tissue, or mucous membranes leads to thickening and induration. Occurring in the intermuscular cellular planes, it may bind down the muscles and tendons, and prevent their free contraction, and may similarly lead to adhesions between joint-surfaces and in the pleural and peritoneal cavities. When occurring in glandular organs, the secreting cells are destroyed, and the function of the organ is proportionately impaired. Organisation in or round the walls of tubes or ducts causes obstruction of the lumen, leading to stricture, or the formation of retention-cysts. In the nervous centres the most serious results may follow if the nervous elements are replaced by new connective tissue.

As in acute, so in chronic inflammation, the process may terminate in death of the tissues leading to ulceration or abscess. Chronic abscess may steadily progress until it bursts, or the fluid parts may be absorbed and a caseous mass result; this may subsequently calcify, and remain permanently harmless, or excite further mischief at a later date (see p. 48).

Signs and symptoms.—The signs of chronic inflammation are similar in nature to those of the acute form, but are necessarily modified in degree. The colour is dusky, livid, and cyanotic, in place of the bright red of acute inflammation; pigmentation is common, owing to changes occurring in the hæmoglobin set free from the escaped red cells. Swelling is the most characteristic sign; it is never very great, and the swollen parts are dense, hard, and brawny from cellular exudate and fibroid overgrowth. The local heat is not perceptibly increased, nor does the patient complain of it.

Pain is less acute, and has not the throbbing character of acute inflammation. It is rather a sense of aching, tenderness, and indefinite neuralgia. In chronic inflammation of dense fibrous structures, or of bone, the pain may be very severe. In some cases pain is caused by implication of the nerve fibrils in the fibroid tissue, and if there is loss of substance, *e.g.* ulceration, the nerves may be exposed, and cause acute pain. The function of a chronically inflamed part is slightly lessened in proportion to the extent of the mischief.

The constitutional symptoms of chronic inflammation are really those of the dyscrasia to which it is due. In simple cases there are none at all. If suppuration accompanies the process, there may be nocturnal elevation of the temperature by one or two degrees; and as soon as the abscess is opened, hectic fever may result if the pus decomposes. The leading symptoms are, in the majority of cases, dependent upon the damage inflicted on the organ by the fibroid induration.

Treatment of chronic inflammation.—General treatment.—As in many cases chronic inflammation is the expression of some general condition, *e.g.* syphilis, the constitutional treatment is of primary importance, and must be that which is applicable to the dyscrasia present.

In all cases the general health and hygienic surroundings must be attended to—tonics, good food, cod-liver oil, maltine, etc., being given. Small doses of mercury, sometimes combined with the iodides of ammonia, potash, or soda, are often serviceable in removing inflammatory induration in cases not necessarily dependent upon syphilis. Arsenic, strychnine, iron, quinine, and vegetable bitters are also useful. Calcium sulphide is strongly recommended by some in chronic tubercular affections, but its value is open to question. Change of air and scene and residence in some watering-place are also to be recommended.

Local treatment.—Any local exciting cause must of course be

removed, such removal being often all the treatment necessary; but in many cases this cannot be done, and means must be adopted to combat the inflammation. Local bleeding and the application of heat are seldom used. The main object is generally the improvement of local nutrition by encouraging the circulation, and hence unloading the congested vessels. The principal means of effecting this are rest, the elevated position, careful bandaging or strapping, cold douching and massage. In chronic ulceration due to varicose veins, these should be tied. In some cases the employment of counter-irritants is very serviceable; they presumably act by drawing an increased supply of good blood to the part, and hence favouring nutrition.

Tincture of iodine is a favourite but almost useless application; if used it must be made to blister the skin, and this can be more readily done by liquor epispasticus. Blistering no doubt gives much relief in some cases, *e.g.* chronic neuritis, and materially helps absorption of inflammatory fluid as in hydrarthrosis; but in many cases blisters are useless. When used, they must be frequently repeated.

The actual cautery applied to the superficial parts of the skin is useful in chronic neuritis, arthritis, and some rheumatic affections. Free incision into the inflamed tissues frequently does much good, especially in chronic inflammation of bone accompanied by pain.

Chronic inflammation of mucous surfaces is best treated by the application of astringent antiseptic lotions.

If suppuration occurs, the treatment is that of chronic abscess (see p. 49).

CATARRHAL INFLAMMATION

Catarrhal inflammation attacks mucous membranes, the process occurring in the sub-epithelial structures. The epithelium itself resists destruction (unless the process be very severe, or the irritant kills it outright), while the vessels beneath dilate, and the exudate is poured into the tissues. Moderate degrees of irritation, while powerful enough to excite inflammation of the sub-epithelial structures, merely stimulate the epithelium itself, the cells of which multiply with great rapidity and are cast off in large quantities. Some of these cells contain leucocytes or micro-organisms. The escaped leucocytes pass to the surface between the epithelial cells, so that there may be definite purulent catarrh. In very severe

inflammation the superficial structures are killed and ulceration ensues. The surface of the mucous membrane is deeply congested and injected with blood; the secretion is increased, and mixed with it are leucocytes, mucus, and desquamated epithelium cells in various stages of degeneration. There is some swelling, but little pain. If the process becomes chronic, there is considerable thickening, pigmentation, and ulceration in patches.

Treatment.—The discharges must be washed away with antiseptic astringent lotions, such as boric acid, Condy's fluid, alum, or chlorate of potash. Special treatment may be required according to the seat and cause of the mischief, *e.g.* bladder, throat, etc.

CHAPTER III

Suppuration and Abscess

Under certain circumstances inflammation may terminate in death of the exudate and liquefaction of the tissues, with the formation of pus. Suppuration may be acute or chronic, localised or diffused, superficial or deep. When destructive inflammation occurs in a wound or on the surface of the skin or mucous membranes, the discharge escapes and appreciable portions of tissue may die *en masse*; this will be further considered under Ulceration and Sloughing (see chap. iv. p. 56). When pus forms in the deeper tissues it may be diffused, or enclosed in a cavity forming an abscess.

Acute suppuration—acute abscess

Etiology.—Acute suppuration is dependent, probably in all cases, upon the presence and action of pyogenic organisms, coupled with a certain predisposition of the tissues which enables these organisms to thrive. Pyogenic organisms may be present in the blood or on the surface of the skin or mucous membranes without inducing suppuration, and indeed they are constantly so present both on the skin and mucous membranes and in our surroundings.

Suppuration then is dependent on (*a*) a predisposition of the tissues, (*b*) the action of organisms; but the relative part played by these two factors is not always the same. If the dose of the poison is large, or the virulence great, suppuration will ensue in tissues but little predisposed to its occurrence; but if the tissue-resistance is feeble, a small dose, or organisms of attenuated virulence, will induce a like result.

A. **Predisposition of the tissues** is brought about by any condition lowering the vitality upon which their power of resistance to

irritants depends. Enfeebled health from advancing years, chronic alcoholism, disease of the *primæ viæ*, especially of the kidneys, or some constitutional dyscrasia diminish the vitality of the body generally. Local resistance may be impaired by heat, cold, chemical irritation, tension, mechanical injury, or inflammation. Subcutaneous injury may lead to inflammation or cause rupture of small vessels and thereby admit into the tissues, where they find conditions favourable to growth and development, any organisms which may be circulating in the blood.

Open wounds offer a ready means of ingress for organisms which may be at hand, and, if the dose be strong enough, these will excite suppuration.

B. **The influence of pyogenic organisms.**—Organisms are

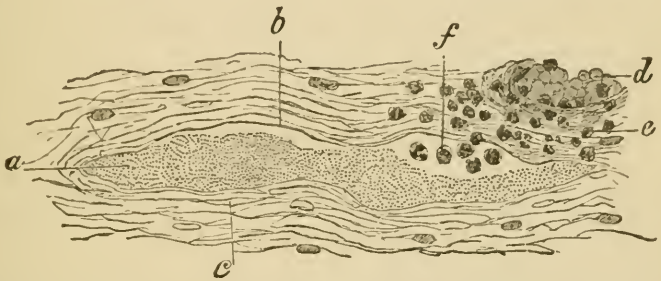

FIG. 7.—Colony of streptococcus erysipelatis (Ziegler). *a*, streptococci within a lymph vessel *b* grouped together partly in globular masses and partly in chaplets like torulæ; *c*, tissue round the lymph vessel with pale, non-staining nuclei; *d*, vein; *e*, cellular infiltration; *f*, cells within a lymph vessel.

present in the pus of all acute suppurations, and many experiments have shown that their presence is not merely accidental, but is the actual cause of the process. Garré induced suppuration and large carbuncular patches on his arm by inunction of gelatine cultivations of staphylococcus pyogenes aureus.

Bockhardt, Bumm, and many others have similarly demonstrated the pyogenic properties of this and other organisms, and in each case the organisms were found in the pus of the abscesses which formed. All pyogenic organisms have not the same degree of virulence, nor has any special kind the same virulence under all circumstances; nor, again, are they all equally common in acute suppuration, as will presently be shown.

Pyogenic organisms are abundantly present everywhere, and may gain entrance to the body by wounds, abrasions, or by the ducts of the skin or glands opening on the surface (*e.g.* the breast); they may also enter the blood by the mucous surfaces, especially if these be

abraded or inflamed. The organisms may spread by the lymphatics (Fig. 7, p. 39) or by the blood-stream; when present in the blood they may be destroyed and excreted by the kidneys and do no harm; but should they meet with tissues whose vitality is impaired, suppuration will ensue. In some cases, masses of cocci are arrested as emboli in the capillaries (Fig. 8) and (rest favouring their growth and activity) local points of suppuration ensue, as is seen in the formation of secondary abscesses in pyæmic infection. Anything favouring rest and lodgment of the organisms in the vessels or tissues enables them to act effectually; thus, injury accompanied by extravasation of blood containing organisms may lead to suppuration, and parts in which the circulation is naturally feeble are favourable seats of invasion. More than one form of organism may be present (mixed infection), and in some cases at least the virulence of the poison is thereby increased; thus, experiment has shown that acute necrosis in rabbits is more severe if staphylococcus pyogenes aureus and albus are present together, than if either is alone, the dose in each case remaining the same. All pyogenic organisms possess in a high degree the power of peptonising albumen, and hence they bring about liquefaction of the tissues and prevent coagulation of the inflammatory exudate; they also give rise to toxines, to the absorption of which the constitutional symptoms are due.

FIG. 8.—Colonies of micrococci within the hepatic capillaries. (Ziegler.)

Pyogenic organisms.—*Staphylococcus pyogenes aureus* is the most common. It is abundant everywhere, and is found in more than 70 per cent of cases of acute suppuration. It is present in acute abscesses, boils, carbuncles, osteomyelitis, infective periostitis, ulcerative endocarditis, septic infection, etc. It liquefies gelatine, and in the presence of air the colonies assume an orange colour.

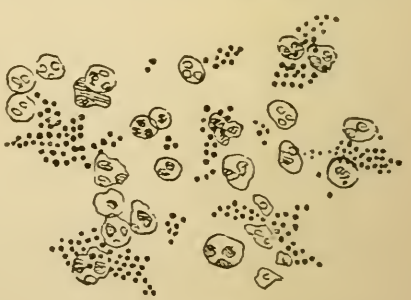

FIG. 9.—Staphylococcus pyogenes aureus and pus cells.

Staphylococcus pyogenes albus is similar to staphylococcus pyogenes aureus, with the exception of the colour, and is met with in the

same cases. Its virulence is probably less, and it is not so common.

Streptococcus pyogenes is described by some as occurring in different forms, varying in their virulence and the products of their activity. These organisms set up diffuse inflammation, which spreads by the lymphatics, whereas the staphylococci more frequently cause circumscribed acute abscess. Streptococci are met with in spreading gangrene, cellulitis, septic infection, and inflammations of an erysipelatoid nature. The virulence of the organisms is great.

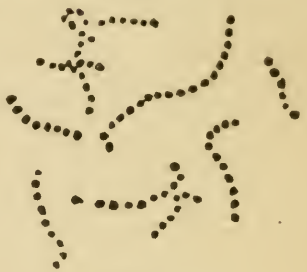

FIG. 10.
Streptococcus pyogenes.

Streptococcus erysipelatis is, according to some, identical with the preceding streptococcus; but other authorities consider it a distinct species (see p. 122).

The foregoing are by far the most common organisms causing acute suppuration; but others, of less virulence or of more limited distribution, are met with.

Diplococcus gonorrhœæ (see Gonorrhœa, p. 156).

Diplococcus pneumoniæ is met with in pneumonia, empyema, and occasionally in cerebral and other abscesses.

Staphylococcus pyogenes fœtidus is present in abscesses containing putrid pus, *e.g.* intestinal or cerebral, and those in connection with mucous membranes.

Bacillus coli communis is constantly present in the intestine and fæces, and is met with in cases of peritonitis and suppuration in the neighbourhood of the intestines.

Staphylococcus cereus albus, *staphylococcus cereus flavus*, *staphylococcus pyogenes citreus*, *micrococcus tenuis*, and some others are occasionally met with.

Mode of action of pyogenic organisms — Formation of abscess.—Pyogenic organisms which have gained entrance into the tissues, or have lodged in a small capillary, exert a powerful peptonising action on the cells in their neighbourhood, in consequence of which the parts undergo coagulation-necrosis, and, losing their structure, present a hyaline, homogeneous appearance (Fig. 11, p. 42). Surrounding this necrotic zone, the tissues, irritated by the products of the organisms—though in less degree—become acutely inflamed, and are speedily infiltrated by leucocytes and inflammatory exudation. Very soon the leucocytes invade the zone of necrosis; the organisms,

freely multiplying, pass into the inflamed area, kill the leucocytes, and prevent coagulation of the exudate by their peptonising properties. Thus a small abscess is formed (Fig. 11), or, if the organism is virulent enough or spreads by the lymphatics, diffuse suppuration follows. Limitation of the process—as in acute abscess—is due to the fact that the cocci at last meet with tissues whose resisting

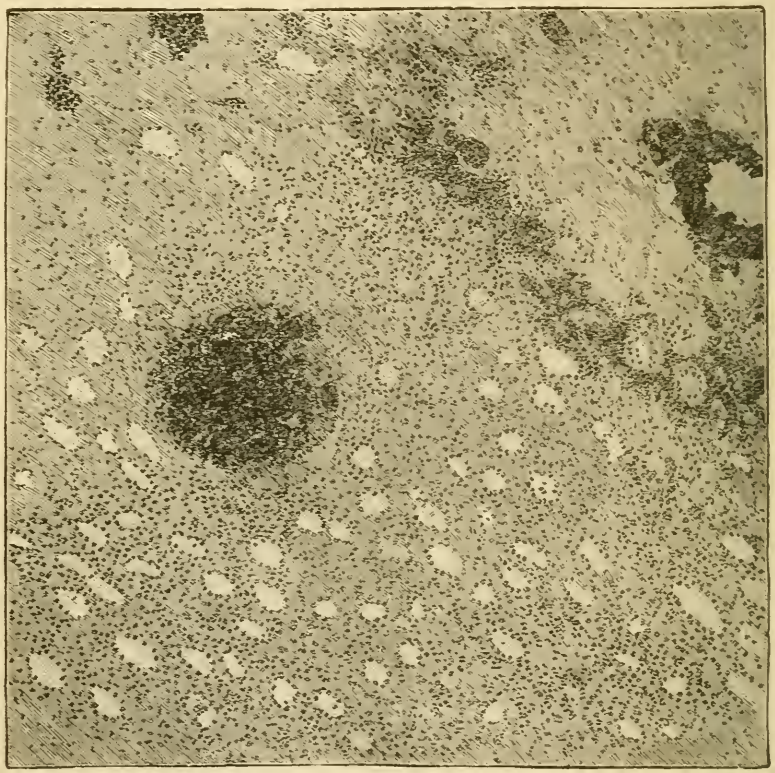

FIG. 11.—Section of kidney, showing in the upper corner a mass of micrococci, a clear necrotic ring, and a layer of inflammation. In the centre is the further stage of the process; the inflammatory cells and the micrococci have now infiltrated the necrotic ring, and an abscess is the result. (Watson Cheyne.)

powers are capable of checking their destructive properties, and hence the pus is surrounded by a layer of granulations and leucocytes. An abscess once formed enlarges through liquefaction of the neighbouring tissues, by a process similar to that to which it owed its origin. Certain tissues, *e.g.* fibrous tissue and blood-vessels of any size, resist the destructive action, and consequently purulent collections may be subdivided by bands of undestroyed tissue.

The wall of an abscess is infiltrated with coagulated lymph and

leucocytes; the small vessels in the destroyed area are inflamed, thrombosed, and subsequently destroyed; larger vessels escape destruction. Abscesses spread in the direction of least resistance, and ultimately burst at the weakest spot.

When the pus has escaped, the cavity of the abscess is considerably diminished by the elasticity and contraction of the tissues, and ultimately—provided circumstances are favourable—is closed by the development of granulations.

Pus is a thick, viscid, alkaline fluid of a pale yellow colour, with a sp. gr. of 1030. It consists of 80 per cent of water, the remainder being albumen, salts (chiefly sodium chloride), leucin, tyrosin, cholesterin, fatty matter, and cellular débris. On standing, the pus cells (*i.e.* dead leucocytes) sink to the bottom, and the liquor puris (*i.e.* non-coagulated fluid exudate) forms a lighter top layer. Sometimes it is blood-stained (sanious pus), the colour imparted being dependent on the amount of blood and the time it has been shed. In cases of suppuration round a blood-clot the mixture is rusty-coloured, from degenerative changes in the red cells. If the corpuscles are few in number, the fluid is thin and watery (ichorous pus), while in other cases the fatty matter and disintegrated pus cells form small masses combined with fibrinous material, giving the pus a curdy appearance.

The pus in abscesses connected with secreting organs may be mixed with the normal secretion; thus, in liver abscesses it is of a characteristic reddish-yellow or green tint, from admixture of bile. After exposure to the air, the colour of pus may alter according to the colour-producing properties of the organism present—blue pus, from the presence of the bacillus pyocyaneus, is only met with outside the body.

Pus has normally a faint, sweetish odour; but it may be offensive from the presence of putrefactive organisms. The pus from some cerebral abscesses, from empyemata, and from those in connection with mucous surfaces, is intensely fœtid, from the presence of the staphylococcus pyogenes fœtidus.

After removal from the body, pus readily decomposes, as it will do when stagnant in ill-drained abscess cavities. Decomposition is, however, impossible in unopened or unaspirated abscesses, since putrefactive organisms can only gain admission from without (p. 94).

Pus corpuscles are dead leucocytes, but a few still living may be occasionally found; they are rounded in shape, and coarsely granular, with a lobed or tripartite nucleus, which is rendered clear by the addition of acetic acid. Much granular detritus (especially in

chronic cases) is often present, owing to disintegration of the pus cells.

Many of the pus cells contain organisms which are also found free in the fluid.

Local signs of acute abscess.—All the signs of acute inflammation are present in a pronounced degree, but are somewhat altered. The pain is often very severe, and of a throbbing character. The swelling, on the advent of suppuration, rapidly increases, provided the distensibility of the part permits; it is brawny and œdematous from the presence of fluid exudate in the surrounding tissues. The presence of œdema, in cases of acute inflammation where suppuration is likely to occur, is very significant of the presence of pus, and often leads the surgeon to operate even in the absence of fluctuation. This is especially the case in deep-seated suppuration. The tense, brawny swelling soon softens at one spot, the softened area being surrounded by a more or less evident ring of dense infiltrated tissue. This area gradually increases, and the abscess approaches the surface, or "points," and, if left alone, soon bursts. The skin over an abscess is of a dusky purplish hue, but as the pus comes to the surface the colour fades, and the skin becomes tense, shiny, and glazed. Abscesses in connection with the gut are often tympanitic, from the presence of gas.

If the fingers are placed over an abscess, and slight pressure is made at a point opposite with the other hand, a wave is felt—fluctuation. This may be difficult or impossible of detection in deep-seated abscesses, especially if they are very small; the surgeon must then rely on the other signs, notably the œdema, for his diagnosis. It should be remembered that fluctuation ought never to be felt for in a direction across the long axis of muscles, for here a sensation very like it can always be obtained. Abscesses may give rise, on account of their position, to special symptoms, *e.g.* cerebral, perineal, retro-pharyngeal.

Constitutional symptoms of acute suppuration.—The constitutional symptoms are dependent on the absorption of toxines formed by the organisms, and are merely an exaggeration of those met with in acute inflammations generally (see p. 26). Suppuration is usually ushered in by a feeling of chilliness, sometimes by shivering fits, or even a definite rigor, especially if an important organ is the seat of mischief. The temperature rises and assumes a remittent type, showing a variation of perhaps 3° or 4° F. As soon as the abscess is evacuated, the local and general symptoms quickly subside, provided free drainage be ensured and putrefaction prevented by

antiseptics. Should putrefaction occur, granulation and healing are arrested, and the patient is exposed to the risk of septic absorption.

Diagnosis of acute abscess.—This is usually readily made, but it has occasionally happened that an inflamed aneurism has been mistaken for abscess. A knowledge of the history, and a careful examination of the case, will usually be sufficient to avoid error (see chap. ii. vol. iii.).

Treatment of acute abscess.—As soon as suppuration has occurred, no time should be lost in giving free exit to the pus. Delay in opening an abscess is not only useless, but may be dangerous, as it may burst into some cavity or hollow viscus, thereby entailing the most serious consequences; moreover, in all cases the abscess goes on increasing in size until it has been evacuated. The incision should be long enough to ensure free drainage, and should be so placed that it is opposite the most dependent part of the abscess, provided the anatomy of the parts will permit. In planning the incision, it must be borne in mind that asepticity is of paramount importance, and the opening should be made in such a position, when possible, as to ensure the least risk of contamination from without. When an abscess has been incised, its cavity should be explored by the finger, and any fibrous septa which may be found subdividing it should, if necessary, be broken down, so that there may be no "pocketing" of discharge; the presence of loculi may necessitate the establishment of counter-openings. As a rule, an abscess cavity does not require washing out, but if the pus is fœtid, *e.g.* in abscesses in connection with the gut, gentle syringing with an unirritating antiseptic lotion should be employed for a few days, until the discharge becomes sweet. For this purpose, one drachm of tincture of iodine to the pint of warm water, or four grains of boric acid to the ounce, are the best solutions; sterilised water may also be used. A small quantity of iodoform emulsion, injected into and allowed to remain in the cavity, is a powerful deodoriser. Solutions of mercury or carbolic acid should be avoided in the case of large abscesses, or, if used, must be very weak, for they may be absorbed by the granulations and give rise to toxic symptoms. The cavity of an abscess must be efficiently drained by a good-sized tube, introduced—if necessary by a pair of sinus forceps—to within about an inch of its deepest part; the calibre of the tube must be of good size, to allow the fluid to escape. As healing takes place the tube will be gradually pushed out of the wound, and must be shortened from time to time, but should not be dis-

pensed with until the wound is quite superficial. If the opening be large, a flanged tube is the best, as its shape prevents it from slipping into the cavity; in other cases the tube may be retained in position by threads of silk or by a safety-pin so placed that it lies across the long axis of the incision.

All abscesses must be dressed antiseptically, according to the method described in chapter i. vol. ii. The dressing must be changed frequently if there is much discharge, but as healing progresses it may be left undisturbed for a longer time; at first most abscesses require dressing once in every twenty-four hours, large ones more frequently. Should the discharge soak through the dressing, this must be immediately changed or packed. When an abscess has been opened and there is much surrounding inflammation, it may be advisable to foment the parts; boracic fomentations are the safest, and should always be employed on account of their antiseptic properties. Poultices, not being antiseptic, should in all cases be avoided.

Methods of opening abscesses.—The most suitable situation for the incision having been determined, it may be made by cutting from without inwards, or by transfixion with a bistoury or Syme's knife. The latter method has the advantage of rapidity and does not cause so much pain, but it should never be employed in parts where important structures may be damaged. Deep-seated abscesses, or those situated in dangerous regions, should be opened by Hilton's method. A small incision is made through the superficial structures, and the cavity is then reached by insinuating a pair of sinus forceps through the deeper tissues; as soon as the abscess has been reached, pus escapes by the side of the forceps, whose onward progress is no longer resisted; the forceps are then opened and withdrawn, and the wound, being stretched apart, may subsequently be enlarged by the fingers or by incision with a blunt-pointed bistoury.

Acute abscesses of an infective nature, *e.g.* bubo, should be carefully and thoroughly sharp-spooned when they have been opened; by this means sloughy tissues are removed, and the cavity, freed of irritating material, heals much more quickly.

ACUTE DIFFUSE SUPPURATION.—See Cellulitis, p. 127.

CHRONIC ABSCESS

Chronic abscesses differ materially from acute in their etiology, course, signs, dangers, and treatment.

Etiology.—The ordinary pyogenic organisms are but rarely found in chronic suppurative processes, which in the vast majority of cases are due to the liquefaction and disintegration of tubercular deposits. The first step in the process is the infiltration of the tissues by tuberculous material which forms a hard, dense mass; the cells undergo caseation, and the broken-down ones, mixed with fluid, form the curdy, watery pus so characteristic of chronic suppuration. By a gradual breaking down of the abscess wall and surrounding tissues, or by the coalescence of neighbouring foci of suppuration, the abscess increases in size. Syphilis is also a cause of chronic suppuration.

When, as sometimes happens, a chronic abscess suddenly assumes subacute or acute characters, the ordinary pyogenic organisms may be found in the pus. Acute abscesses may, if the organisms perish, become chronic.

Anatomy and course.—The progress of a chronic abscess is very slow, and may extend over a period of many months before the patient's attention is directed to it. Chronic abscess is commonly met with in diseases of bones or joints, in the lymphatic glands and subcutaneous tissue, and in organs which are the seat of tubercle. Extension takes place along the lines of least resistance, *e.g.* along connective tissue planes, under the sheaths of muscles, or along arterial tracks. Large abscesses which have burrowed far may present numerous cavities connected together by narrow tracks at the points of greatest resistance; thus, when a psoas abscess passes beneath Poupart's ligament it is contracted, widening again in the thigh. Owing to the chronicity of the process, the abscess wall is usually of considerable thickness through induration of the soft structures, so that it may have a distinctly cystic character, thus enabling the surgeon to dissect it cleanly out—a most important point, since the wall is infiltrated with tuberculous matter which, unless removed, will perpetuate the mischief. The density of the wall may be so great that the abscess simulates a solid tumour.

The walls of chronic tubercular abscesses are infiltrated with tubercle bacilli and nodules, the inflammatory induration gradually diminishing as more healthy parts are reached. Unhealthy granulations line the interior, and masses of coagulated exudate and cells may form a more or less definite lining—the so-called pyogenic membrane of former times.

Arterial trunks in the neighbourhood of chronic abscesses generally escape destruction; but they may be denuded and stretch across

the cavity as rounded cords, and care must be taken not to mistake them for fibrous septa. In pulmonary cavities this condition is frequently seen and is one of danger, since the artery, deprived of its natural support, may become dilated or aneurismal. The bursting of such an aneurism is the commonest cause of fatal hæmoptysis.

Occasionally the pus is like that met with in acute abscess, but in the majority of cases it is thin and watery, and contains masses of broken-down cells and lymph which give it a curdy appearance. In tubercular cases the discharge is infective, though tubercle bacilli are not often found, as they have undergone disintegration, and only the spores remain; if, however, it be injected into guinea-pigs or other susceptible animals, general tuberculosis results.

Sooner or later most chronic abscesses point and burst, but this may not occur for many months or at all. In the latter case the fluid parts of the pus are absorbed and the caseous detritus remains encapsuled, a permanent cure perhaps resulting; or else the caseous patch may, even after many years, again become purulent through slight irritation (Residual Abscess). Lastly, the caseous mass may become infiltrated with lime salts, and the whole shrink and be invested with a fibrous capsule; when this has occurred the calcified mass is practically a foreign body, and may remain quiescent, or, acting as an irritant to the tissues, may be a factor in determining future suppuration in its neighbourhood under other favouring circumstances.

Dangers and complications.—Apart from the dangers and complications which may occur from the presence of a tubercular focus in the body, and those which are inseparable from collections of purulent material in important regions, a chronic abscess, when opened, exposes the patient to the risks attending putrefactive decomposition of the discharges, which is especially liable to occur if the abscess cannot be efficiently drained or asepticity ensured (Chronic Septic Intoxication, p. 108). Moreover, when a chronic abscess has been opened and drained, the constant discharge will exhaust and undermine the health of the patient, and may lead to albuminoid and fatty changes in the viscera. It is of paramount importance that the possibility of these dangers should be borne in mind when determining on the best course of treatment to pursue in any given case. Under ordinary conditions and proper treatment cure may usually be brought about, but there are unfortunately a certain percentage of cases in which the primary mischief (*e.g.* spinal caries) continues to advance in spite of all our skill and attention, and the discharge

persists as copiously as at first, the patient ultimately sinking from exhaustion too often complicated by septic absorption.

Signs and symptoms.—A chronic abscess may exist for a long time without showing evidence of its presence, enlarging so slowly that the parts adapt themselves to the swelling. It is common experience that in cases of spinal caries with psoas abscess the symptoms may be all referred to the diseased vertebræ, the patient being quite unaware of the existence of even a large abscess.

The local signs of acute inflammation are absent, although there may be a little redness and œdema of the skin over the abscess. Pain may or may not be present. In large abscesses distinct fluctuation can be obtained, but in deep-seated and small ones this may be quite absent, the thickness of the walls imparting to the swelling the characters of a solid growth, of which the density varies from elasticity to almost stony hardness. Many cases of chronic breast abscess have only been diagnosed after amputation of the organ for supposed cancer. Chronic abscesses may, by the pressure they induce, lead to special symptoms in different regions of the body, just as would be the case from a solid growth.

Constitutional disturbance is absent so long as the abscess is unopened, and even after this provided no putrefaction occurs. Sometimes there is a slight rise of temperature at night, and this, coupled with other indications, is of great diagnostic value.

Diagnosis.—The diagnosis of chronic abscess is by no means always easy, it being often confounded with a solid or cystic tumour. The chief points to rely upon in arriving at a diagnosis are the presence of fluctuation, œdema of the overlying tissues, want of complete circumscription of the tumour, and the presence of some source of irritation, *e.g.* diseased bone; should there be a nocturnal rise of temperature in conjunction with the other signs, the diagnosis of chronic abscess is usually certain. Solid and cystic tumours are usually more clearly localised and have a well-defined margin, whereas the indurated tissue surrounding a chronic collection of pus fades away gradually as healthy parts are approached. In cases where an absolute diagnosis cannot be made, the tumour may be explored with a fine syringe or aspirator, or by means of an incision; the latter course should always be adopted in doubtful tumours of the breast before resorting to amputation.

Treatment.—The treatment of chronic abscess depends in great measure on its origin and seat, and in considering diseases of special organs and tissues the methods to be adopted will be indicated.

Simply emptying an abscess by aspiration is merely a temporary

means to relieve tension; the pus rapidly reaccumulates and cure cannot be hoped for. The persistence of chronic abscess is usually dependent on a tubercular condition of its walls, and so long as this state remains, no useful end is gained by drawing off the purulent collection.

Aspiration with the injection of iodoform emulsion offers a better chance of success than does aspiration alone. The iodoform exerts a beneficial action on the abscess wall, and may arrest the morbid process and bring about a more healthy state. Sometimes one injection suffices for cure; more often the fluid reaccumulates, though in less quantity, and the operation must be repeated. It may completely fail, and should not be relied on except in cases unsuitable for one of the following methods.

Subcutaneous and glandular abscesses may be dissected out entire, like a simple cystic tumour. The wall should be completely removed and the wound closed, union by first intention resulting in most cases. This is the ideal treatment of chronic abscess, and should be adopted wherever practicable; the patient is completely freed of his disease and a healthy wound left.

Some subcutaneous and glandular abscesses may not, on account of their position and connections, be capable of complete removal by excision. In such cases the sac should be freely incised, the pus evacuated, and the entire wall dissected away with the knife or snipped away with scissors. If any doubt remains as to the complete removal of all the diseased tissue, the cavity should be well sharp-spooned and treated with chloride of zinc (40 grs. ad ℥i.) or iodoform emulsion. The wound may now be closed and union by first intention will usually occur. Sometimes the cavity fills again and the operation has to be repeated. In cases where, even after dissection and scraping, it is probable that some of the diseased tissue still remains, no attempt should be made to close the wound; it should be well iodoformed and packed with strips of antiseptic gauze. These may, under ordinary circumstances, remain untouched for three or four days; on their removal the interior of the abscess will be found covered with healthy granulations, but should any part still show evidence of disease, it will require sharp-spooning again.

In cases unsuited for treatment by the above means, *e.g.* spinal abscesses, a small incision should be made at the most suitable spot and the pus evacuated. The wall of the abscess is then freely sharp-spooned with a flushing gouge, the detritus being washed away by sterilised water or weak boric acid solution. The scraping should be complete and continued until no more detritus comes

away, due care being taken not to perforate the abscess wall. The cavity should now be completely emptied and dried with rough sponges, so as to remove all débris. Iodoform emulsion is applied to all parts of the wall, and a little left in the cavity. The wound is carefully closed and dressed, the dressings remaining undisturbed until union is complete. A drainage tube should usually be employed for a couple of days to remove the serous fluid which will be poured out. This treatment may at once bring about a cure, but in some cases the abscess fills again, the fluid being more watery and not so abundant. Under such circumstances the operation must be repeated, and this done a third or even a fourth time if necessary. In successful cases the cavity is obliterated and the abscess is represented by a fibrous mass, often with a caseous centre. Sometimes failure results, the wound breaking down and the pus escaping externally; cure may even then be brought about by a repetition of the process, the edges of the wound being freely sharp-spooned and then carefully approximated.

If in spite of every care a chronic abscess cannot be made to heal but threatens to burst, the surgeon is compelled to lay it open and drain it. The incision must be large enough to ensure perfect drainage, and be so placed that the risk of putrefaction is reduced to a minimum.

If a chronic abscess is dependent upon disease of a bone or any similar condition, that disease must itself receive appropriate treatment; thus, sequestra must be removed and carious cavities scraped.

The importance of strict asepsis in chronic abscess is paramount. If putrefactive organisms gain admission, they not only give rise to toxines which, being absorbed, produce septic intoxication, but by their local action they impart a fresh impetus to the disease. No one is justified in opening a chronic abscess unless he is perfectly familiar with the details of antiseptic surgery, and can ensure perfect asepticity.

The constitutional treatment is directed chiefly to building up the patient's health. Good food, tonics, malt liquor and stimulants if necessary, with plenty of fresh air, are essential.

Sinus and Fistula

SINUS

When an abscess has been opened or has burst, it may not completely heal, a narrow suppurating canal or sinus persisting.

Anatomy of sinuses.—A sinus is a narrow channel, often of great length, sometimes straight, but more usually sinuous, opening externally by a narrow mouth; the deeper portion may, on account of insufficient outlet, be dilated by retained discharge into a small abscess cavity (Fig. 12, *C*, p. 54). The walls in old-standing cases are tough and indurated; the inner surface varies in appearance according to the age of the sinus. When recent, the granulations are numerous, though pale, flabby, and unhealthy; they bleed readily on probing, and are highly sensitive; in chronic cases the inner surface may be smooth and almost devoid of granulations, and probing does not cause pain or bleeding unless roughly performed. Sinuses may be multiple, as in cases of diseased bone.

The external opening of a sinus is usually its narrowest part, and is often—especially in sinuses of recent formation, and those due to bone disease—surrounded by exuberant, unhealthy granulations. The purulent discharge is unhealthy and watery in nature, and may contain lime salts if the sinus leads to diseased bone. In some cases the discharge is copious, in others scanty; in all it is likely to be fœtid from decomposition. The mouth of a sinus may be so narrow that it becomes temporarily occluded either by congestion or by the formation of a scab, the pus, accumulating behind, gives rise to symptoms of abscess formation; but when tension is high enough it escapes, only to re-collect.

Causes of sinus.—An abscess may remain unhealed from one or more of the following causes, but is much more likely to result in the case of chronic abscess (so often dependent on tubercle) than in that of acute:—

(1) The opening is too small, or not in the most favourable situation, so that the discharge does not have free exit.
(2) Putrefaction of the discharge.
(3) The presence of a foreign body, *e.g.* sequestrum.
(4) Want of rest owing to its situation among muscles.
(5) Its specific nature, *e.g.* tubercle; the virus being present in the granulations and in the wall of the sinus, does not allow healing to occur.
(6) General ill-health of the patient.

Diagnosis.—The diagnosis of the existence of a sinus is usually simple, but sometimes the opening is so small and so well concealed among folds of tissue, *e.g.* round the anus, that careful search is necessary. The chief point is to determine the course and origin of the suppurating track. It must be remembered that abscesses

travel in the direction of least resistance, and that their point of bursting by no means necessarily corresponds to that of their origin, *e.g.* spinal abscesses. The history of the case often affords much information, but chief reliance must be placed on careful and gentle probing; gentleness is necessary, not only to lessen pain, but to avoid pushing the probe through the wall of the sinus into the surrounding tissues, and so being misled.

The appearance of a sinus is sometimes suggestive of its origin; thus, in the case of caries, the mouth of the sinus is swollen and filled with pale unhealthy granulations, while in necrosis the latter are absent and the mouth is depressed. The situation is sometimes useful in determining the seat of original mischief; for instance, in hip disease originating in the acetabulum the sinuses are usually present about the pubic and gluteal regions and Poupart's ligament; but when the synovial membrane or head of the femur is the primary seat of the mischief, the sinuses open in the vicinity of the great trochanter.

Treatment.—A knowledge of the causes leading to the establishment of a sinus indicates the steps necessary for effecting a cure.

An inefficient opening must be enlarged so that free drainage is ensured, and one or more counter-openings may be necessary. Any foreign body, such as a sequestrum, must be removed. If the interior of the sinus is unhealthy, either by reason of neglect, putrefaction, or the specific nature of the mischief (*e.g.* tubercle), the sinus should be freely laid open and "sharp-spooned," or the wall dissected out, and the wound having been rendered aseptic, should be allowed to granulate from the bottom. If a sinus can be completely dissected out, the wound may be sutured and healing by first intention secured. Sometimes the position of a sinus among important structures does not admit of such radical treatment, and the surgeon must be content with enlarging the orifice and attempting to induce healthy action by means of stimulating and aseptic lotions, such as red wash (zinc sulph. grs. 2, tr. lavander ℥ 10, aq. ad ℥i.) or chloride of zinc, gr. 1 to ℥i. distilled water. A probe, warmed and coated with nitrate of silver, and passed down the sinus, occasionally induces healing.

If movement of neighbouring muscles keeps up constant irritation, this must be counteracted by rest, position, and such other means as seem appropriate to the case. If the general health is feeble, good food, tonics, and country air are the chief indications.

FISTULA

A fistula is practically a sinus opening into some cavity or hollow viscus and on to the surface of the skin (Fig. 12, *A*). Fistulous openings, without any external orifice (Fig. 12, *B*), may also exist between cavities or tubes, *e.g.* vesico-vaginal fistula.

Anatomy of fistula.—Fistulæ usually present similar characters to those described under sinus. They may be single or multiple, and vary considerably in length and in the course they take, so that the external opening by no means necessarily corresponds with the seat of the original mischief. A fistulous track may not open directly into the cavity with which it communicates, but may burrow along the soft structures. This is well seen in some cases of fæcal fistula resulting from gangrene of a strangulated hernia.

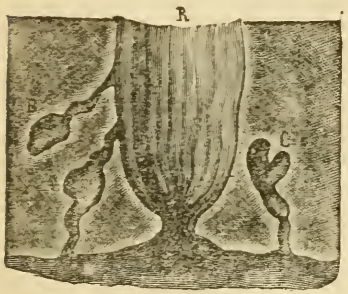

FIG. 12.—Diagram of the common forms of fistulæ *in ano*. *R*, rectum; *A*, complete fistula, dilated in the middle; *B*, blind internal fistula; *C*, blind external fistula, the sinus dividing at the upper end. (Foliin.)

Old fistulous communications between two mucous surfaces, *e.g.* vesico-vaginal, may not show evidence of the cause to which they owe their formation, the margins of mucous membrane being soundly healed, and the track itself lined with epithelium.

Causes of fistula.—A fistula may result from suppuration, the abscess bursting externally and into some cavity, or from injury which either at once establishes the communication or, at a later date, causes it through sloughing. Fistulæ are not infrequently formed as the result of invasion by and breaking down of a malignant growth, *e.g.* entero-vesical. Fistulous openings are sometimes made by the surgeon for the relief of strictures and other conditions, *e.g.* tracheotomy, colotomy. Lastly, fistulæ may be due to some congenital defect, *e.g.* branchial fistulæ.

In whatever way a fistula is formed, it is kept open by the passage along it of the contents of the tube with which it communicates, and the question as to whether it will heal or not depends in great measure upon the permeability of the canal into which it opens. Thus, perineal fistulæ occurring as the result of stricture of the urethra will not heal unless the stricture be treated, whereas a similar fistula intentionally made for the purpose of exploring the bladder heals readily under ordinary circumstances.

Diagnosis of fistula.—When a fistula has a direct opening on

the surface, the character of the discharge is sufficient evidence of the nature of the case; thus, urine escapes through perineal, fæces through intestinal fistulæ.

If there is no external opening the diagnosis is more difficult. Thus, a fistula opening into the rectum, but not externally, must be sought for by the speculum; while an entero-vesical fistula may be diagnosed by the presence in the urine of undigested particles of food, and sometimes by the passage of flatus *per urethram*.

Treatment.—If a fistula communicates with a tube, the lumen of which is contracted, the stricture must first be dilated or cut. As soon as there is a free passage along the canal, the fistula will, if of recent formation, usually heal soundly and quickly, provided it is kept clean. Healing may be hastened by the passage of a hot wire along the track of the fistula, or it may be laid open and allowed to heal from the bottom, as in fistula *in ano*.

If a fistula remains unhealed, and if it be due to sloughing of the soft parts, its treatment depends chiefly on the size of the artificial communication, and on the state of the walls. Small openings may be made to close by introducing a fine cautery point as already stated; but large ones require some form of plastic operation suitable to the case. No such operation should, however, be undertaken until all inflammation has been arrested for some time.

Fistulæ due to the invasion of cancerous or sacromatous growths need no treatment beyond the observance of cleanliness. Congenital fistulæ, if amenable to treatment, must be dissected out, since the walls are lined with epithelium which, unless removed, effectually prevents closure.

CHAPTER IV

Ulceration and Ulcers

Inflammation causing death of the skin or of a mucous surface gives rise to ulceration. If portions of tissue, large or small, die *en masse*, they are called "sloughs," and the process is known as "sloughing," and stands, as it were, in mid-position between ulceration and gangrene. When ulceration is the outcome of inflammation of a non-specific character, it is said to be simple. Specific ulcers are those dependent on the action of a specific virus, *e.g.* soft chancre, tubercle, syphilis. Cancerous or sarcomatous tumours may invade and destroy superficial structures, and an open wound, or so-called malignant ulcer, results; the term ulceration cannot properly be applied to such cases.

Simple or Non-specific Ulceration

Etiology.—The **predisposing causes** of ulceration are those local or general states which, by diminishing the resisting power of the tissues, favour their death as a result of inflammation. A knowledge of these causes is of primary importance, not only in the prevention, but in the cure of ulceration. Among the *general causes* —all of which act by lowering vitality—may be mentioned old age, insufficient and improper food, bad hygiene, chronic alcoholism, and certain constitutional dyscrasiæ, such as gout, scurvy, diabetes, syphilis, and tubercle; and not only do these diseases favour the occurrence of simple ulceration, but the formation of ulcers of definite clinical characters is a manifestation of the diseases themselves.

Local causes act by interfering with the due nutrition of the part, thus rendering it very intolerant of even slight irritation. The most potent of these is certainly chronic congestion from impeded venous

return. A striking example of this is to be seen in ulceration of the leg as the result of varicose veins, especially in those who stand for many hours during the day (Fig. 13). In the presence of venous congestion, the slightest scratch may be sufficient to determine the commencement of ulceration which may persist for years. Thrombosis of the veins leads to a like result. After phlegmasia dolens, ulceration of the legs is by no means uncommon; and so long as the circulation is imperfect, the ulcers prove very refractory to treatment, breaking down again and again on the slightest provocation. Deficient arterial supply through disease of, or pressure on the vessels, defective innervation of the tissues, pressure, or continued mechanical or chemical irritation, may all induce ulceration. Previously inflamed parts and cicatrices, of which the vascular supply is poor, are liable to ulcerate from slight causes.

FIG. 13.—Varicose ulcer (*a*) with varicose veins of the leg. (Tillmans.)

The **determining causes** are practically identical with those of inflammation, acting with slight intensity, and for a considerable time. Mechanical irritation, *e.g.* vesical calculus or badly fitting surgical appliances, may induce ulceration or sloughing, according to the degree of damage inflicted. Chemical irritants, even of feeble action, may have similar results in congested tissues, or in patients whose general health is disordered.

Specific poisons, introduced locally through a small abrasion, or present in the blood (*e.g.* syphilis), give rise to special forms of ulceration to be subsequently referred to.

In the case of a simple callous ulcer, many causes usually act together in bringing about the process. These ulcers are usually met with among the poor of large cities—patients, therefore, ill-fed, ill-clad, and who are too frequently surrounded by faulty hygienic arrangements, or are the subjects of chronic alcoholism. A slight scratch,—say on a leg, which is the seat of varicose veins,—hardly

noticed at the time, gradually enlarges until a definite small wound is produced; this is probably neglected, and, above all, not kept clean, and speedily develops into a chronic ulcer. In such a case the exciting cause is insignificant, but the attendant local and general conditions, all tending to lower vitality, culminate in a state of things which would otherwise be quite inconsistent with its trivial origin.

Ulceration of mucous surfaces is usually of a specific nature, but may be induced by mechanical irritation, *e.g.* ulcers of the tongue from sharp teeth or badly-fitting tooth-plates.

Morbid anatomy.—When inflammation occurs in the skin or mucous membranes, and terminates in death of the tissues, the débris, mixed with fluid, escapes from the surface as discharge. In consequence of inflammation, the superficial vessels are engorged and many are thrombosed; leucocytes and fluid exudate pervade the area of inflammation, infiltrating the tissues, and penetrating among the epithelium cells, which proliferate and are cast off. Deprived of their blood-supply, the superficial parts and numberless leucocytes perish and undergo molecular disintegration. If the thrombosis be extensive and the irritant exciting these changes acts with marked intensity, large or small masses of tissue die *en masse*, slough, and are gradually separated by the phagocytic action of the leucocytes. An ulcer increases in size so long as its causes remain operative; inflammation and thrombosis continue at the margins and in the base of the sore, which gradually increases in area and depth. The rapidity of the destructive process depends chiefly on the nature of the irritant; in the case of simple ulceration it is usually slow, unless putrefaction occurs, but if any infective organism is present, destruction may spread with alarming rapidity, *e.g.* hospital gangrene. Large vessels in the vicinity of an ulcer usually escape destruction, except in cases of an infective and rapidly-spreading nature. The smaller vessels are obliterated by thrombosis, and are destroyed with the other tissues. Occasionally even chronic ulcers, *e.g.* gastric, may open arteries, and give rise to profuse or fatal hæmorrhage. Again, varicose ulcers may destroy the wall of an enlarged vein.

As ulceration extends in depth, neighbouring structures may be injuriously affected; thus, chronic ulcers of the leg not uncommonly give rise to periostitis (Fig. 14, p. 59), or even to caries or necrosis. Ulceration on mucous surfaces of hollow organs may, especially if the process be acute, lead to perforations, *e.g.* typhoid ulceration; but in chronic cases adhesions form between the base of the ulcer

and neighbouring parts, and perforation is thereby prevented, *e.g.* chronic gastric ulcer. As soon as the causes inducing ulceration have ceased to act, destruction comes to an end, and granulations spring up and continue to grow until the surface is reached, when healing—materially aided by contraction—is completed by growth of the marginal epithelium. The appearance of the base, margins, and tissues surrounding the ulcer differ according to circumstances, and will be stated under each form.

FIG. 14.—Osteoplastic periostitis of the tibia. In the centre is a smooth area which formed the floor of an ulcer; round this the new bone forms definite nodules (Westminster Hospital Museum, No. 155). (Drawn by C. H. Freeman.)

General principles of treatment.—**Constitutional measures,** calculated to improve the general health and restore lost recuperative power, must be adopted. Any dyscrasia which may be present, although itself not the actual cause of the ulceration, must receive its appropriate treatment.

Among the poor, simple ulceration is too often evidence of bad and improper feeding, and defective sanitation. In such cases these defects must be remedied as far as the patient's circumstances will permit. Cod-liver oil, malt extract, the vegetable bitters, quinine and iron should be given in cases of anæmia and general debility. Opium, in grain doses twice a day, is a most valuable remedy in some cases. Under its use, inflamed, sloughy, and indolent ulcers often undergo rapid improvement.

Local measures. — All local sources of irritation must be removed, and every endeavour made to subdue inflammation, arrest the destructive process, and bring about granulation. Rest and elevation are important in all cases.

Antiseptics.—It is imperative that the surface be rendered aseptic, more especially in those ulcers dependent on specific organisms. The presence of putrefactive organisms not only prevents granulation, but, on account of the irritation produced, favours the spread of the ulcer. The parts round the wound should be thoroughly cleaned with soap and water, and all fatty and sebaceous material removed by ether and ammonia; unless this be thoroughly done, reinfection of the ulcer will take place. The surface of the sore should be

cleansed by hot boracic fomentations, which also favour the separation of any sloughs; or the surface may be freely swabbed over with pure carbolic acid, or a strong solution of it. Chloride of zinc (40 grs. ad ℥i.) is an efficient antiseptic, and is by some preferred to carbolic acid. Asepsis may also be secured by dressing the sore for a few days with carbolic acid solution (5 per cent), or 1 : 1000 or 1 : 2000 mercuric solution. When putrefaction has been arrested by any of these means, reinfection must be prevented by the use of mild, unirritating antiseptic dressings, *e.g.* boracic acid.

When a chronic ulcer has been thoroughly cleaned, the pain so often attending it is materially diminished. In the case of infective ulceration, more energetic measures than those indicated are required to get rid of the irritating organisms (see Hospital Gangrene, Sloughing Phagedæna, pp. 116, 118).

Restoration of the circulation and relief of venous engorgement is essential in all cases. Circulation may be encouraged by gentle friction and massage of the limb, by rest and elevation, by compression with bandages or strapping, and, in the case of varicose veins, by operation, if the general circumstances of the case permit of its performance (see chap. i. vol. iii.).

Compression by bandages or strapping must be equable, otherwise more harm than good will result. It should be applied from the extremity to some distance beyond the ulcer—as far, indeed, as the circulation is impaired. When the ulcer has been dressed in the manner to be presently described, an ordinary surgical or Martin's elastic bandage must be carefully and evenly applied, tightly enough to give support without obstructing the veins. It should be put on before rising in the morning, and may be dispensed with at night. A clean thin woollen stocking worn beneath the bandage and changed every day serves to absorb the discharge and makes the patient more comfortable. Martin's bandage is liable to set up eczema, as it prevents evaporation; this, however, is less likely to occur if it be perforated. The bandage should be lightly applied, as it becomes tighter when the patient walks about and the limb fills with blood. Martin's bandage must be kept thoroughly clean, and dried before reapplication, and must not be used with any greasy dressing.

Strapping with adhesive plaster is an excellent means of compression. It must be reapplied every day, or oftener, according to the amount of discharge. Compression should, when possible, be combined with rest and elevation.

Dressings applied to ulcers must be antiseptic, unirritating, free from grease, and must be changed sufficiently often to ensure perfect

cleanliness. Antiseptic moist warmth, in the form of boracic fomentations, combined with rest and elevation, is most useful in the early stages of ulceration, in chronic cases with adherent and decomposing sloughs, and in all inflamed and sloughy states. The fomentations should be thick, large enough to cover a considerable extent of the surrounding parts, and changed every four hours or oftener. When, by their use, inflammation has subsided, the sloughs have separated, and the sore has been brought into a healthy state, it must be carefully dressed with some form of lotion.

Boracic acid (4 grs. ad ℥i.) is the best; it keeps the surface clean, but does not cause any irritation. Red wash (zinc sulph. grs. 2, tr. lavand. co. ♏ 10, aq. ad ℥i.) is also useful, while lead and opium lotion may be used if there is much pain. Great care is needed in the application of a dressing. The surface and surrounding parts should be gently syringed with some of the lotion to remove all discharge, it should then be lightly covered with a piece of lint wetted with the solution, which should only cover the granulating surface; for if it be too large, the growing epithelium at the edges is macerated, and healing retarded. Over the lint a piece of protective is placed, and should project about one-eighth of an inch beyond, this again is covered with a thin layer of salicylic wool, and the whole retained in position by a bandage lightly and evenly applied from below upwards. The dressing should be changed each day, or oftener if there is much discharge.

Ointments, spread on butter-cloth, are much used by some; but they have the disadvantage of being greasy, and not easily removed. They should be made with vaseline, paraffin, or lanolin, and never with ordinary lard, which rapidly putrefies. Boracic ointment is the most useful.

When the granulating surface is weak, the granulations being flabby, œdematous, and exuberant, it should be stimulated with red wash, or lightly touched with a stick of silver nitrate.

Dr. Findlater, of Edgware, has been very successful in treating large chronic ulcers by the following plan:—The ulcer is covered with a piece of coarse muslin or leno (which has been cleansed and softened in boracic or carbolic solution) applied from below up as a bandage, and made to exert firm pressure. This is not removed for a fortnight, and may remain untouched even longer. Outside it a piece of lint, saturated with weak boracic solution or red wash, is applied, covered with oiled silk and kept in position by a few turns of a bandage; this outside dressing is reapplied once or twice daily according to the amount of discharge. I have given this method

some attention, and have been surprised with the excellent results obtained, especially after first grafting the ulcer (Figs. 15, 16). The resulting scar, after skin grafting and treatment by this method, closely resembles true skin, and is certainly superior to that obtained by any other. The discharge from the ulcer oozes through the meshes of the muslin, and must be washed away each time the outer dressing is changed. If there is much discharge, the meshes of the muslin must be coarse. The muslin exercises equable compression on the edges and base of the ulcer, and has the advantages of strapping without confining the discharge. The ulcer may, if clean enough, be first grafted. I have had many opportunities of appreciating the success of this plan in cases of ulcer under my care, and have been much struck with the rapidity with which healing has occurred, even in those of the most unpromising nature.

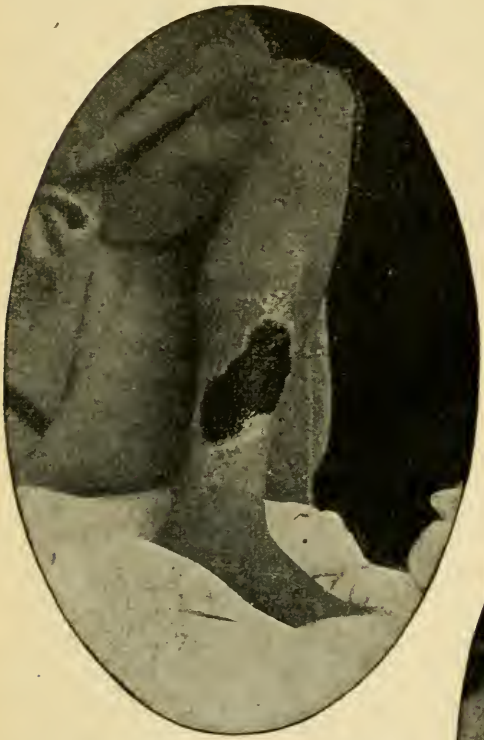

FIG. 15.—A chronic ulcer of the leg of thirty years' duration in a man æt. fifty-one. (Photograph by Mr. Budd.)

FIG. 16.—The same leg as Fig. 15, taken thirteen days after skin grafting. The grafts were protected by Findlater's method, and the dressing was left untouched for ten days. The scar subsequently became quite sound and closely resembled normal skin in appearance. (Photograph by Mr. Budd.)

Dr. Findlater also uses this method in the treatment of burns.

The oxygen treatment, introduced by Dr. Stoker, has in his hands proved very successful. The part on which the ulcer is situated is isolated in a chamber containing an atmosphere of pure air charged with 40 per cent oxygen. Ozone is said to give better results. The treatment is continuous, and takes a long time to effect healing, but should be tried when other means fail—doubtless the enforced and prolonged rest does much good.

Skin grafting.—Healing may be considerably hastened, and a sounder scar with diminished contraction ensured, by skin grafting. The grafts may be taken from the patient, or from another person. In the latter case, every care must be taken to select a perfectly healthy individual; but the practice is objectionable, and has been known to convey syphilis to the patient (p. 170).

Reverdin's method—Epithelial grafts.—The ulcer must be thoroughly clean, and the surface (usually the arm or thigh) from which the grafts are to be taken must be rendered aseptic. Minute portions of epidermis are shaved off or snipped off with a fine pair of scissors curved on the flat, while the skin is caught up in a fine pair of forceps; these are then laid on the ulcer, the several grafts being placed about half an inch apart. A piece of protective cleansed in carbolic or boracic solution is placed over all, and a light dressing applied and left undisturbed for three or four days, when the situation of those grafts which have "taken" will be indicated by a slight depression, though they may not be distinguishable, since the opaque superficial cells will probably have desquamated. In a day or two, however, small white areas, surrounded by blue and pink zones (epithelial islets), are distinctly visible. The new epithelium spreads over the surface and neighbouring islets coalesce. Such a scar is not very stable, and may break down from slight causes.

Thiersch's method produces more rapid healing, a more stable scar, and minimises the contraction of the ulcer. It may be advantageously employed in operations where the edges of the wound cannot be approximated, and in plastic surgery.

Strict asepsis and a healthy state of the wound and patient are essential.

The granulations and healing edge of the ulcer, if unhealthy, must be scraped away or shaved off with a razor, so that the firm young fibrous tissue at its base is exposed. A piece of clean protective is then placed on the denuded surface, and over this sponges are firmly bandaged to arrest all hæmorrhage. It is not, however,

by any means necessary to scrape away the granulations provided they be clean and healthy, for the grafts will grow on them perfectly well.

The grafts are most conveniently taken from the inner side and front of the thigh. A broad flat razor, wetted with boracic solution, or .5 per cent salt solution, to prevent them sticking, is made by a gentle sawing movement to cut large grafts, including about half the thickness of the skin, which is put on the stretch by an assistant. This just divides the tops of the papillæ, and causes slight oozing from the cut surface. The grafts tend to curl up, and may be best applied by floating them from the razor on to the surface, on which they are placed raw-surface downwards. The several grafts should slightly overlap, and the whole sore be completely covered. The curled-up margins may be straightened out by a probe and forceps. The wound is then covered with protective, which has been cleaned in boracic solution. Outside this a layer of salicylic or boracic wool is applied, and the whole dressing firmly bandaged on and left undisturbed for five or six days, when the grafts will be found to have taken. The muslin bandage, as described on p. 61, is an excellent means of keeping the grafts in position and ensuring rest. Complete rest and freedom from all sources of local irritation must be observed for some weeks, or else the scar may break down. The wounds left by removal of the grafts must be covered with boracic ointment or iodo-vaseline, and left untouched for six or seven days, when healing will have occurred.

Varieties of simple ulcer.—The healthy or healing sore is nothing more than a granulating wound. The outline varies in shape, but is usually circular or ovoid; the surrounding tissues have lost all trace of inflammation, but some of the vessels may be hyperæmic, and there may be slight staining. The edge is not indurated, but is regular, gradually bevelled towards the surface, and presents three zones distinguished by their colour. The innermost zone is composed of a single layer of epithelium cells, advancing over the granulating surface from the margin; this zone is red, and is surrounded by a thicker layer of cells of a bluish tint, the colour being due to the absence of the horny cells of the epidermis. Externally, where the epithelium cells form a thick layer and the horny cells are present, the colour is dead white; this zone may be considerably increased in area by maceration of the superficial cells if wet dressings are applied. The base of a healthy sore is quite supple, and its contraction accelerates the healing process. The

surface is level and covered with florid granulations, and here and there may be seen undestroyed islets of epithelium. The discharge is very scanty and is pure pus. There is no pain, even if the surface of the sore be touched.

Indolent or callous ulcers are usually met with in the lower third of the leg in patients beyond middle life, especially in association with varicose veins. The base and edges are hard and indurated; the latter are steep-cut, irregular, and raised, and show no signs of epithelial proliferation. Induration is due to the development of new fibrous tissue which acts detrimentally in two ways: (1) by preventing contraction, and (2) by narrowing the vessels and preventing the due supply of blood. The induration extends for some distance beyond the ulcer, and the tissues are often congested and hyperæmic. The base is considerably below the level of the edge; it is adherent to subjacent parts and covered by unhealthy granulations exuding a sanious, purulent discharge. The indolent ulcer is very chronic and may completely surround the limb, being then often incurable (annular ulcer). It is generally painless, but may cause much suffering, either as the result of implication of the nerve endings in the connective tissue, or, in the case of the leg, from periostitis of the bone beneath (Fig. 14, p. 59).

Treatment.—The surface of the ulcer and parts around must be rendered aseptic, and an attempt made to get rid of the induration and depress the edges. This may be accomplished by rest, elevation, or massage, and especially by bandaging or strapping. Strapping exerts equable compression, thereby encouraging the circulation and promoting absorption of the effused lymph in the indurated edges and base of the ulcer. Findlater's plan of treatment is very useful (Figs. 15 and 16, p. 62). Some surgeons strongly recommend stimulation of the surrounding congested parts by means of blisters. When the surface assumes a more healthy appearance, stimulating lotions, *e.g.* red wash, should be applied, or it may be occasionally touched with silver nitrate. Opium internally is often of great use, and, occurring as these ulcers so frequently do in the poor and half-starved, every effort must be made to ensure better living and more healthy surroundings, treatment which is unfortunately more easily recommended than followed. Indolent ulcers extending completely round the limb may necessitate amputation.

The weak ulcer is covered with exuberant pale, flabby, gelatinous granulations which project beyond the surface, and are apt to slough and break down on slight irritation. Such a condition is not uncommonly seen in debilitated patients.

Treatment.—This form of ulcer requires stimulating with some astringent lotion, such as red wash, or by touching with the solid nitrate of silver. Rest, elevation, and strapping are also necessary. If these measures fail, the surface should be sharp-spooned.

The irritable ulcer is most usually met with at the lower third of the leg or behind the outer ankle. It is especially prone to occur in women, and has many of the characters of an indolent ulcer. The surface is often in a dirty sloughing condition, discharging a small quantity of sanious pus.

The pain may be extreme, especially at night; it is dependent on exposure of the nerve endings, and to their irritation by the chemical products of putrefaction, and also to their involvement in the new fibrous tissue.

Treatment.—Free scraping of the surface or the application of strong carbolic or nitric acid is indicated. In cases of severe pain a tenotome may be passed beneath the base of the ulcer, which is then freed from its deep connections and the nerves divided. If the pain can be located at certain points by running a probe over the surface of the ulcer, it may be stopped by passing a fine knife under such tender areas as recommended by Hilton. In less severe cases lead and opium lotions may be effectual. The internal administration of opium is often necessary and most beneficial.

Varicose ulcer.—Ulcers dependent on the presence of varicose veins are so named (Fig. 13, p. 57). They present the ordinary characters of the indolent ulcer (p. 65).

Treatment.—The varicose ulcer must be treated on the same lines as the indolent. If circumstances permit, the varicose veins should be operated on; at the same time the surface of the ulcer is sharp-spooned, thoroughly cleansed, and dressed antiseptically. When the dressings are removed at the end of a week or ten days the surface will be found granulating healthily, and in the case of a small ulcer practically healed

Hæmorrhagic ulcer is spoken of in cases where hæmorrhage from the granulations and into the base of the ulcer give it a purplish colour. The hæmorrhage sometimes occurs at the menstrual period, and the general appearance of the sore is sometimes suggestive of sarcoma.

Treatment.—As these ulcers usually occur in anæmic women they demand the exhibition of iron and tonics coupled with good food. Locally, bandaging or strapping is beneficial. Their occasional dependence on scurvy should be borne in mind with regard to treatment.

Inflamed ulcer.—In some cases simple ulcers may be accompanied by considerable acute inflammation, with its local and constitutional symptoms. The destruction of tissue is rapid and the discharge copious, often mixed with blood and shreds of slough. Putrefaction is common. Inflamed ulcers are chiefly met with in drunkards and persons of bad constitution.

The sloughing ulcer is an exaggerated form of the inflamed, and is usually, though not always, of a specific nature (see Sloughing Phagedæna and Hospital Gangrene, pp. 118, 116).

Treatment.—Inflamed and sloughing ulcers must be treated by absolute rest and elevation of the part, which should be continuously fomented with hot boracic acid. If there is much exudation into the surrounding tissues, incisions may become necessary for the relief of tension.

SPECIFIC ULCERS DEPENDENT ON SOME CONSTITUTIONAL DYSCRASIA

Ulceration forms one of the clinical features of some diathetic states. Such ulcers present certain peculiarities of appearance, situation, and progress which readily distinguish them from each other and from simple ulcers.

Ulceration of mucous membranes is usually of a specific nature.

Syphilitic ulcers, see p. 184.

Tubercular ulcers, see p. 152.

Scorbutic ulcers are accompanied by the usual manifestations of scurvy and are caused by the most trivial injuries or scratches, or originate in a scar. They are often seen about the mouth and may cause extensive sloughing of the gums and mucous membrane. Scorbutic ulcers are of a livid colour. The surrounding tissues are swollen and œdematous and the ulcer tends to spread. The surface is covered with unhealthy granulations and exudes a fœtid sanious discharge which often forms a thick spongy crust, removal of which excites free bleeding. The crust rapidly re-forms.

Treatment.—The ulcerated surface must be kept clean and free from all sources of irritation. The general treatment is that of scurvy, for which the reader is referred to a work on medicine.

Diabetic ulcers.—Diabetic patients are very prone to sloughy ulceration, often from very slight causes, *e.g.* blistering. A dark-coloured slough may form, which gradually extends in depth and area. Putrefaction is common unless cleanliness be observed, and

the process shows a tendency to spread and very little towards healing. Pain is usually severe, but may be absent (see p. 85).

Treatment.—Unirritating antiseptic lotions should be freely used and the sore treated on the general principles laid down (p. 59). Opium must be given in large doses and the general disease treated.

Gouty ulcers.—The subjects of gout are specially prone to chronic congestion and inflammation, and not infrequently develop very indolent superficial ulcers about the lower third of the leg. Such ulcers are often surrounded by eczematous skin (*eczematous ulcer*). The discharge is thin, and often contains urate of soda. In some cases the skin inflames over a "chalk-stone," and a deep ulcer results, which constantly discharges masses of the salt.

Gouty ulcers are very persistent, remain stationary for a long time, and tend to recur again and again from the most trivial causes.

Treatment.—Rest, with the application of a mild unirritating lotion, such as boracic acid, is the best local treatment. The diet must be carefully regulated, beer and wine being interdicted, and the quantity of meat and pastry limited. Saline aperients, colchicum and citrate of potash, should be prescribed.

ULCERS DEPENDENT ON LESIONS OF THE NERVOUS SYSTEM

Certain ulcerations may occur in connection with wounds of nervous trunks and in diseases of the central nervous system, with which they will be described.

ULCERATION AND SLOUGHING DEPENDENT ON LOCAL INFECTION

Inoculation with certain micro-organisms may lead to extensive sloughing of the tissues, perhaps more closely allied to gangrene than to ulceration. These conditions will be described in chap. vii.

ULCERS DEPENDENT ON THE PRESENCE OF TUMOURS

Cancer and sarcoma may gradually infiltrate and destroy the skin so that the substance of the tumour is exposed. Any tumour may cause ulceration of the skin over it as the result of stretching, or from pressure on the vessels. Malignant tumours may, when exposed, fungate on the surface; or the ulceration may extend deeply into their substance and cause a large, crateriform, foul ulcer (Fig. 62, p. 254).

Soft growths are more likely to fungate. In any case the growth of the tumour more than makes up for the loss by ulceration, so that it continues to increase in size.

Rodent ulcer is a special form of epithelioma, slow destruction of all tissues in its neighbourhood being the chief clinical feature.

For further information, see pp. 232, 253.

CHAPTER V

Gangrene or Mortification

Death of a portion of the body without previous disintegration of its structure is known as Gangrene or Sloughing, the latter term being usually reserved for death of small areas only; the dead portion is known as a *slough* or *sphacelus*. Gangrene may occur in any part of the body, and under such different conditions, that the effects and symptoms it produces vary within the widest limits.

Etiology.—In most cases of gangrene several causes combine to bring about the death of the part; some are adjuvants only, but all have this in common—they lower the vitality of the part, in the majority of cases, by interfering with the circulation through it. Thus, the lessened vitality and cardiac weakness of old age, coupled with arterial degeneration leading to thrombosis and complete occlusion of the vessels, are the factors in the production of senile gangrene.

The predisposing causes are those which, by lowering general or local vital activity, favour the occurrence of gangrene in the presence of exciting causes. Old age, cardiac insufficiency, general disease of the arterial system, deficient nervous influence, renal disease, diabetes, and many of the acute fevers are predisposing causes of gangrene which, under such conditions, may occur from quite trivial causes, *e.g.* a small wound.

Determining causes. — *Simple acute inflammation* of healthy tissues very rarely results in gangrene, but it may do so if from any cause, *e.g.* diabetes, nutrition is affected. Inflammation dependent on certain micro-organisms may be so intense, and accompanied by so much exudation, that the vessels are compressed, and gangrene results, *e.g.* gangrene of the skin in cellulitis; or the corrosive action of the chemical products of the organisms may kill the

tissues, *e.g.* hospital gangrene. Thus the causes of inflammation are also causative factors in some cases, but not in all, for the process may occur independently of any antecedent inflammatory condition.

Severe mechanical (Fig. 19, p. 75) *and chemical injuries and extremes of heat and cold* induce gangrene, either directly in consequence of the actual damage done to the tissues or vessels, or secondarily, as a result of inflammation, the vitality of the parts having been seriously impaired by the injury. A part severely crushed is killed outright; and even if the larger vessels are not ruptured, circulation through them is impeded, and the blood is no longer capable of passing along them owing to the damaged state of their walls.

FIG. 17.—Gangrene of the finger resulting from poisoning by the sting of a weaver fish (*Trachinus draco*, Linn.). The poisonous mucus is lodged in a deep, double groove in the spines of the dorsal fin and opercular spines. These fish are dreaded by fishermen on account of the dangers they attribute to a wound from one of the spines which they cut off before handling the fish. Swelling rapidly extends to the shoulder, accompanied by pain. Fishermen say that smart friction with oil soon restores the part to a normal condition. See *Fishes of the British Islands*, by Couch. (Drawn by C. H. Freeman.)

Pressure on the tissues is primarily exercised at the expense of the fluids, viz. the blood and lymph. The first effect of injurious pressure will be felt by the thin-walled veins; the circulation through them is impeded, and œdema results, thereby increasing the pressure, and leading to arterial obstruction. The ill-effects of pressure are specially noticeable in patients in bad general health, or in parts which from any cause are not properly nourished, *e.g.* bed-sores in old and feeble people and on paralysed limbs.

Tension is a form of pressure, and plays an important part in gangrene resulting from acute inflammation; thus, in acute cellulitis the tension of the inflammatory exudate compresses the blood-vessels running to the skin, which consequently sloughs (p. 129).

In obstruction to the circulation the impediment may be in the veins or arteries, and may be due to a variety of causes. From whatever cause arising, diminution in the quantity of blood passing along a vessel is compensated for by the establishment of collateral circulation; should this be insufficient, the effects will depend upon the extent to which the circulation is embarrassed; if the impediment is great, gangrene must result.

Interference with the circulation through the veins may be due

to pressure from without, injury, or thrombosis. Obstruction of an artery is more serious than is that of a vein, because the venous channels are more numerous and larger than the arterial, and if gangrene is to follow venous obstruction many important vessels must be implicated. It is highly probable that gangrene from venous obstruction, independently of arterial, does not occur.

The position of arterial obstruction is a matter of importance; thus, if the popliteal be occluded at its upper part, the safety of the limb is imperilled owing to the scanty collateral circulation about the knee; whereas obstruction of the femoral at Scarpa's triangle is readily compensated for through the profunda and vessels at the back of the limb.

Obstruction to the arterial supply may be due:—

(1) *To causes acting from without, e.g.* ligature or pressure.

(2) *To causes from within, e.g.* thrombosis or embolism.

(3) *To causes acting on the walls, e.g.* injury, calcification, obliterative arteritis.

(4) *To arterial spasm*, as in Raynaud's disease (Fig. 20, p. 83) and ergot gangrene.

(5) *To a mixture of these conditions.*

Defective innervation of the tissues may be an exciting as well as a predisposing cause of mortification; the gangrenous patches which form so quickly and extend so deeply in cases of injury to the spinal cord, and the sloughing of the cornea after injury of the fifth cranial nerve, are well-known instances. Raynaud's disease is probably due to central lesions of the cord inducing vaso-motor spasm; and diabetic gangrene is sometimes dependent on peripheral neuritis. The mode in which nervous influences, or their withdrawal, maintain or diminish the vitality of the tissues is at present undetermined, and it is more than probable that gangrene dependent on injury of nervous matter is of complex production. The existence of trophic nerves—filaments directly responsible, as it were, for the due nourishment of the tissue elements—is at present, although believed in by most, not proved; but assuming such to exist, it is easy to understand how their damage would result in necrosis of the parts they supply. Those who deny, or at any rate doubt, the existence of such direct trophic influence, attribute the gangrene following nervous lesions to vaso-motor paralysis and consequent dilatation of the vessels leading to chronic congestion and impaired circulation; this phenomenon doubtless occurs, and contributes to the occurrence of gangrene, but is in all likelihood contributory only.

In Raynaud's disease and in ergotism, irritation of the sensory

nerves induces a condition of vaso-motor spasm limiting the supply of blood, and such limitation, if persistent or frequently repeated, terminates in gangrene.

Specific micro-organisms may induce acute inflammation terminating in death. This result is partly due to the peptonising action of the organism, and partly to the tension exercised by the inflammatory exudate. Gangrene of this class is of the spreading variety and highly dangerous. The products of putrefaction are capable of inducing extensive sloughing of the tissues with which they come in contact.

Varieties.—Gangrene induced by causes acting directly on the dead part, *e.g.* mechanical injury, cold, is said to be *direct*. When the cause acts at a distance, *e.g.* circulatory obstruction, the gangrene is said to be *indirect*. *Constitutional* gangrene is due to some general condition, *e.g.* diabetes. *Inflammatory* gangrene is the outcome of antecedent inflammation either of a simple or specific nature.

Dry gangrene is usually seen in peripheral parts in which there is little moisture; and as this is lost by evaporation, the dead part becomes mummified.

Moist gangrene occurs in all internal tissues, and in cases where evaporation of fluids does not occur.

General local condition of gangrenous parts.—The general appearance of gangrenous tissues varies according to whether the gangrene be dry or moist; but in all cases the signs diagnostic of death are the same.

Sensibility is completely lost in dead tissues. When gangrene is threatened there is often a feeling of weight, numbness or coldness of the part, and sometimes acute tingling or burning pain is present. The presence or absence of pain, and its degree, depend on the rapidity of the process and the integrity or otherwise of the nervous supply. Pain in a gangrenous part may be merely referred to it in the same way that a patient after amputation of the leg may refer pain to the toes; it may also be due to the continued activity of the nervous elements which have retained their vitality longer than less important structures.

Loss of heat necessarily follows on the death of any part, its temperature then depending on that of its surroundings.

Loss of function.—So long as any muscular power remains in a gangrenous part movement is possible. Thus, in spreading traumatic gangrene, a disease especially spreading along cellular planes, movement of the limb is easy since the muscles are unaffected.

The colour of gangrenous parts usually varies from greenish-brown to black, and is due to the formation of sulphide of iron produced by the action of sulphuretted hydrogen on the iron of the red corpuscles. If the amount of blood in the part be very small, as in cases of arterial obstruction, the colour is often at first of a tallowy, dead white; sooner or later, however, the dead part becomes flecked by streaks and maculæ of brown and black which increase in size and number and fuse, and so change the colour of the whole part.

The appearance as regards swelling, etc., depends upon whether the gangrene is dry or moist.

Dry gangrene (Fig. 18).—The dead part, losing fluid by evaporation, becomes hard and horny, shrivels up, and closely resembles the tissues of a mummy. Swelling and decomposition are absent, and no blebs form on the surface, which has a peculiar greasy feel, and exhales a musty odour.

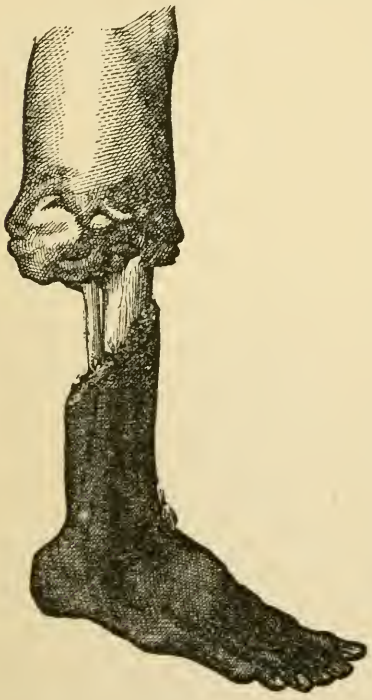

FIG. 18.—Dry gangrene of the foot and leg. (Follin.)

Moist gangrene differs materially from dry, the difference being due to the presence of fluid in the tissues and consequent putrefactive changes. The part quickly swells, undergoes rapid decomposition, and becomes emphysematous from accumulation of gases in the tissues. Blebs containing dark brown offensive fluid form on the surface, and the softened cuticle may be readily detached by merely drawing the finger over the part. The blebs met with in gangrenous parts are always loose and contain coloured fluid, in contrast to those formed as the result of burns, friction, or galling, which are tense and usually contain clear serum, though this may be blood-stained. The surface is often ecchymosed, and the superficial veins may be clearly distinguishable as dark lines. The skin sloughs, and the discharges are horribly offensive. The still living tissues which are adjacent to the dead are swollen from inflammatory exudate, and the lymphatic vessels and glands may be enlarged and inflamed.

The constitutional symptoms of gangrene vary, within the widest limits, according to the extent and seat of the process, the causes inducing it, and the age and general health of the patient. In many cases the condition is a purely local one, and there are practically no general symptoms, *e.g.* frost-bite; in others, *e.g.* gangrene of the bowel or from the action of micro-organisms, they may be of the severest form:—shock, collapse, nervous prostration, and the supervention of typhoid symptoms ushering in a fatal termination.

Separation of gangrenous parts.—Provided that the cause inducing gangrene is limited in its action, and that the seat of the process is not such as to quickly cause the patient's death, the dead parts will be gradually separated from the living by suppurative inflammation occurring at their junction. Some forms of gangrene, dependent on specific causes, show no tendency to spontaneous arrest, and gradually spread until the patient succumbs. The process of separation is as follows:—

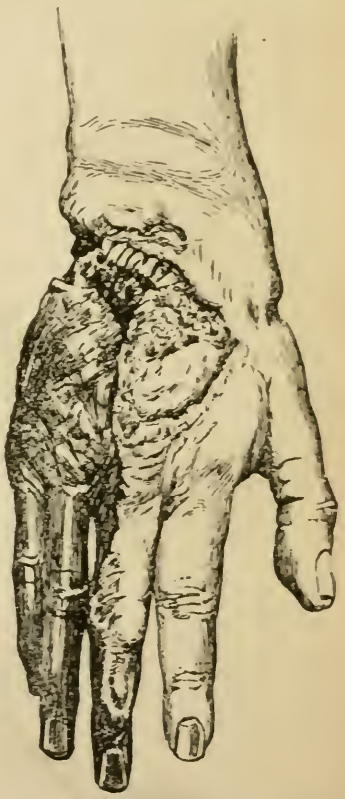

FIG. 19.—Gangrene occurring in a young man as the result of injury. The dead and living parts are separated by a deep line of demarcation. The tendons are seen at the upper part. (Drawn by C. H. Freeman.)

The gangrenous part, acting as an irritant, excites inflammation in the adjacent living tissues. Stasis and thrombosis occur in the vessels, and the junction of the dead and living parts is indicated by a livid, congested line—the "line of demarcation." Leucocytes and lymph infiltrate the tissues, and at the line of demarcation suppuration gradually effects separation. This process of ulceration extends through the entire thickness of the part; but as the gangrene extends higher up in the superficial than in the deep structures, the resulting stump would, if separation were left to nature, be conical (Fig. 18). As separation progresses, the vessels are obliterated by thrombosis, so that bleeding does not usually occur; yet it may be profuse. Tendons, ligaments, bone, etc., prove very resistant.

The time required for the separation of gangrenous tissues varies much; thus, in gangrene of a limited portion of skin, the slough may separate in a few days, while many weeks would be required for the separation of a limb were it left to the unaided powers of nature.

Repair of the surface left after complete separation is brought about by granulation and cicatrisation.

Prognosis in gangrene.—The prognosis as regards life depends upon the extent, situation, and cause of the gangrene. It is always serious at the two extremes of life, and in the debilitated. Gangrene dependent upon strictly local causes of a non-infective nature, and not attacking parts of vital importance, is not dangerous to life in otherwise healthy patients, provided due care be taken to prevent putrefaction, or reduce it to a minimum. Gangrene dependent on some constitutional condition, or upon some specific poison, is always of extreme gravity, indicating as it does severe impairment of the general health, or the presence of a poison of great virulence.

General principles of treatment.—Although each form of gangrene requires special treatment, certain rules are applicable to all. Every endeavour must be made to remove any local or constitutional causes upon which the process is dependent, and the continuance of which favour its spread. The general health must be improved, and the strength maintained, for all forms of gangrene are productive of marked nervous prostration and asthenia.

Constitutional treatment.—Depletion must be avoided; easily digestible food, alcohol, port wine, quinine, preparations of bark, ammonia, and diffusible stimulants being chiefly relied on. Opium is useful in all forms of gangrene, but especially in the diabetic; it allays pain, and, by inducing sleep, gives tone to the nervous system, and seems to have some direct beneficial action in arresting the process. It should be given in grain doses every four or six hours, and the dose may be increased as tolerance is established. Opium must be given carefully, especially in those whose kidneys are diseased, and in the young. The bowels should be kept acting, and the action of the kidneys and skin encouraged. When the process has been arrested and fever has subsided, separation and repair may be hastened by a more liberal diet and tonics.

Local treatment.—The occurrence of gangrene as the result of inflammation may sometimes be prevented by relieving tension by means of free incisions. Injurious pressure must be removed, and the circulation favoured by elevation and warmth.

In dry gangrene all that is necessary is to wrap the part up in a thick layer of antiseptic wool, the ulcerating line of separation being carefully cleaned and treated antiseptically.

In moist gangrene every endeavour must be made to prevent the occurrence of putrefaction and to relieve tension by incisions. Warm antiseptic fomentations are most useful, or the limb may be enveloped in a dressing saturated with some antiseptic lotion, or antiseptic powders may be dusted over it. The most useful antiseptics and deodorants are solutions of carbolic acid, boracic acid, or chloride of zinc, eucalyptus and carbolic oil, and powdered charcoal, boracic acid, or iodoform. Fomentations should be made of boracic lint, or with hot boracic solution.

The part must be dressed with sufficient frequency to ensure cleanliness, but not too often, on account of the pain and distress to the patient. The line of separation must be separately dressed with antiseptic lotions. Sloughs should as a rule be left to separate spontaneously; but should their removal be deemed advisable, in order to diminish the amount of putrefying matter, they may be cut away with scissors, care being taken that still living tissue is not damaged.

Amputation.—In gangrene of the extremities the question of amputation naturally arises. In acute spreading gangrene of an infective nature immediate operation is essential, since no limitation of the process can occur.

In gangrene dependent on local causes, or on arterial obstruction, it is wiser to wait until the line of separation is clearly indicated, so that amputation is performed through tissues of sufficient vitality. In such cases it is impossible to say how much of the limb will perish. Moreover, it is not only necessary that amputation should be performed through still living parts, but their vitality must be sufficiently great to allow of healing, otherwise this may not occur, and sloughing or gangrene of the flaps may result.

GANGRENE DUE TO PRESSURE—BED-SORES

Etiology.—Bed-sores are unhealthy ulcers due to gangrene of the superficial parts as the result of prolonged pressure. In some cases the gangrene is preceded by inflammation, in others it occurs directly from exsanguination. Bed-sores are especially met with in the aged, enfeebled, and cachectic, and in those confined to bed for a long time in consequence of some chronic disease or severe injury. Paralysed parts are very subject to bed-sore, partly on

account of their lowered vitality. Spinal and head injuries, associated with nervous lesions, sometimes occasion deep, rapidly-spreading, gangrenous areas—acute bed-sores. These are probably due to interference with the innervation of the parts, in addition to the usual local causes.

Bad nursing is too frequently responsible for the occurrence of bed-sores; the accumulation of dirt and moisture (*e.g.* urine), infrequent changing of the draw-sheets, and improper bedding being important avoidable causes.

Similar sores may be caused by ill-fitting surgical appliances and splints.

Seat and appearances.—Those parts most subjected to pressure are the favourite situations of bed-sores; they usually form on the sacrum, buttocks, trochanters, malleoli, and heels, more rarely about the scapular regions, and on the elbows and knees. Before a sore has actually formed the skin becomes red and somewhat roughened, and is very tender, except in paralysed and anæsthetic parts. If these indications are neglected—and sometimes even in spite of treatment—a small bleb forms on the surface, gangrene of the skin quickly follows, and is surrounded by a zone of inflammation. In bad cases, especially in the acute sores met with in spinal injury, the gangrene eats deeply into the tissues, exposing and destroying the muscles and bones. The dead and sloughy tissues slowly separate, leaving an unhealthy ulcer, which may gradually extend in area and depth, or exhibit a feeble attempt at granulation. In most cases pain and discomfort are complained of at an early period, and become more severe when gangrene has actually occurred; but in anæsthetic parts, or if the patient be very ill, no such complaint may be made.

Prognosis.—The prognosis in cases of bed-sore depends upon the state of the patient. If the condition which confines him to bed is one inducing serious general disturbance, and if he shows no signs of improvement, it can hardly be expected that the bed-sores will heal, although with careful treatment further damage may be prevented. Bed-sores in paralysed parts are very intractable.

If the patient recovers his general health, these sores rapidly improve. Occasionally they prove fatal from exhaustion, or the supervention of pyæmia. Acute bed-sores, in association with spinal injury, nearly always indicate a fatal termination.

Treatment.—Preventive.—The formation of bed-sores may be prevented in most cases by careful nursing, and the avoidance of

those causes—often trivial in themselves—which have been shown to produce them. The patient should be placed on a comfortable mattress, with a clean, soft draw-sheet immediately underneath him, which should be changed frequently. On no account should he lie on a feather bed or on a blanket; the former rucks, the latter is rough and irritating. The use of a water-bed or water-pillow is the safest preventive measure in very feeble persons. Absolute cleanliness and dryness are essential. The back and other parts liable to pressure should be examined once or twice daily, and, after being washed and dried, dusted with finely powdered boracic acid. If the skin becomes tender or red, it should be rubbed twice daily with spirit or eau-de-cologne, or painted with collodion; or it may be gently rubbed with a mixture of equal parts of balsam of Peru and resin ointment. Pressure should be relieved by frequent change in the patient's position, and by the employment of circular air- or water-cushions.

Curative.—All pressure must immediately be taken off the seat of ulceration. In bad bed-sores on the buttocks or sacrum I have had excellent results from placing the patient on a canvas hammock, with a hole of suitable size cut in it beneath the site of the sore; this effectually relieves all pressure, and allows the necessary dressings to be applied without troubling the patient. If there is merely vesication and separation of the cuticle, the part should be protected by covering it with a thin layer of antiseptic wool and collodion, or by the application of boracic ointment.

When sloughing is fully established, warm irrigation with boracic acid is most useful, or the part may be fomented to encourage separation of the sloughs. As soon as granulation sets in, the wound must be treated like an ordinary healing ulcer.

Constitutionally every effort must be made, by good feeding and stimulants, to improve the general health. Opium is very useful.

GANGRENE FROM ARTERIAL DISEASE—SENILE GANGRENE

Seat.—Senile gangrene attacks peripheral parts in which the circulation is naturally feeble. Most commonly it is seen in the toes or fingers; but the margins of the ears and alæ of the nose are not rarely affected. The gangrene may be quite limited in extent, or may attack many toes or fingers and gradually extend up the limb.

Causes.—Atheroma and primary calcification of the arteries,

combined with cardiac weakness and the general lowered vitality of the tissues through imperfect nutrition, are the essential causes of senile gangrene. Its onset is usually determined by thrombosis in the diseased vessels, or by some trivial injury or inflammation, the nutrition of the tissues being at such a low ebb that bare existence of them is alone possible, and any source of irritation, however slight, results in gangrene.

Signs.—Certain symptoms indicative of impaired circulation are usually complained of before gangrene manifests itself, and their recognition should lead to the adoption of preventive measures. There is a feeling of heaviness in the limb with frequent cramp, tingling or itching, and numbness with perverted sensation, alternating with or accompanied by severe aching pain. The extremity is cold, blue, and cyanosed, and there may be slight swelling, especially at night. All the signs are more marked in cold weather. Pulsation in the arteries is very faint, or may be indistinguishable. In the presence of a wound or slight inflammation, a small slough may form; this dries and shrivels, and the gangrene spreads slowly but surely, and is surrounded by a painful inflamed area, which in its turn dies. In other cases a small ulcer may be the starting-point of the process; or it may begin, in the absence of injury, as a purple or dark spot surrounded by an inflammatory zone. The gangrene is of the dry variety; the dead parts, losing water by evaporation, shrivel up, and resemble the tissues of a mummy. The characters have been already described (p. 74). When more healthy tissues are reached, a line of separation forms, and the dead part may be cast off. Sometimes, even after the process has apparently ceased, the gangrene advances, and a second line of demarcation forms.

Constitutional symptoms may be entirely absent, but if the gangrene is extensive, or there is much pain, the general health suffers. The patient gradually loses health and strength, his nights are broken and sleepless on account of the pain he suffers, and he may succumb from exhaustion. Sometimes there is fever of the asthenic type, especially if there is inflammation and putrefaction at the line of separation.

Prognosis.—The prognosis is always grave, as the occurrence of senile gangrene is dependent on arterial disease and general lowering of vitality. The gravity depends on the extent and rapidity of the process and upon the general state of the patient. Separation of the gangrenous part, if left to nature, may take weeks or months according to its extent.

Treatment.—General.—Depletory measures are of course contra-indicated. The bowels, kidneys, and skin should be kept acting freely by the use of appropriate remedies. The food must be generous in quantity and of an easily digestible nature; it should be given in small quantities at frequent intervals, so as not to overtax the digestive powers. Stout, port wine, and other alcoholic and diffusible stimulants must be given to encourage the action of the heart; the amount being regulated by the state of the pulse. Small doses of strychnine should be prescribed; bark and the mineral acids, cod-liver oil and tonics are useful if the patient can digest them.

No drug is so valuable as opium, and it should be given in gradually increasing doses, beginning with one grain every six hours. Opium gives tone to the nervous system by the relief of pain and by inducing sleep; the improvement it effects is often very marked.

Local.—When gangrene is threatened, the part should be wrapped in cotton wadding and kept in an elevated position, free from all sources of irritation. Gentle friction and tepid bathing may increase the circulation.

When gangrene has actually occurred, the dead parts must be kept dry to avoid putrefaction, and should be wrapped up in antiseptic wool dusted with iodoform or boracic powder. The dressing should not be removed unnecessarily, nor should the part be exposed to the air. The line of separation must be antiseptically dressed and putrefaction prevented; it may be lightly dusted with iodoform and dressed with iodo-vaseline or boracic ointment; wet dressings are better avoided. The whole limb must be elevated and enveloped in flannel or wadding.

As soon as the true line of separation has formed, amputation should be performed before the patient's strength is exhausted. This is always a serious step in view of the general and local conditions upon which the gangrene is dependent; the advisability of the operation and its seat must be determined on the merits of each case. If amputation is performed just above the line of separation, there is a risk of sloughing of the flaps, or of failure in the healing process, since the vitality of the tissues is much diminished; in quite limited gangrene, however, when there is evident pulsation in the vessels above, this operation may be undertaken. In other cases it is wiser to amputate at a distance. Although such a procedure entails more shock, this is counter-balanced by the fact that the parts operated

on are more healthy, and there is a better prospect of speedy cure.

In gangrene of the upper limb, amputation may usually be performed in the middle of the arm; in the lower limb, the knee, or lower third of the thigh are the points of election. In deciding on the seat of amputation, it should be borne in mind that calcification of the arteries is most extensive, and produces most serious effects in the smaller vessels, viz. those below the knee and in the forearm, and if amputation is performed through more healthy vessels, healing will occur more readily. The operation, when decided upon, should not be delayed, and should be done bloodlessly, with the strictest antiseptic precautions. The flaps should not be redundant, nor contain an unnecessary amount of muscle; above all, they must not be scored or injured by the knife. The circular method of operation, giving as it does the least area of raw surface, is the best. The diseased vessels must be carefully secured with carbolised silk, and the dressings should, unless fever or other signs contra-indicate, be left untouched for a week or ten days. Gangrene of the flaps, secondary hæmorrhage, and shock are the chief dangers.

GANGRENES DUE TO ARTERIAL SPASM—SYMMETRICAL GANGRENE—RAYNAUD'S DISEASE

Raynaud's disease is dependent on spasm of the small arteries, probably due to central cord changes. In some cases at least it appears probable that the condition is the result of peripheral neuritis. The spasm induces retardation and arrest of the circulation with venous congestion. These attacks are of short duration, but if frequently repeated may cause gangrene. The disease is usually met with in children, or in women between fifteen and thirty years of age; more rarely it affects males.

Cold, mental perturbation, and local injury may excite an attack.

Signs.—The signs vary in intensity from a mere sensation of numbness to complete gangrene of the fingers. The following stages or degrees of intensity are recognised:—

(*a*) **Local syncope.**—The tip of the finger (or fingers), sometimes in an apparently healthy patient and without evident cause, becomes cold, bloodless, and of a dead white colour; it is anæsthetic and movement is sometimes

lost. The attack may last minutes or hours, and usually recurs at intervals.

(*b*) **Local asphyxia.**—The signs of impeded circulation are more marked. The skin assumes a livid hue, or may appear as if ink-stained, the area of cyanosis being fringed by a vermilion margin. The superficial veins are often clearly marked as livid lines. Motion is diminished or lost, and there is superficial anæsthesia with deep-seated burning pain. The pulse at the wrist is often small and feeble. An attack may, as in the previous state, last minutes or hours, and recurs at intervals; as it passes off, the finger regains its normal colour and is the seat of tingling or stinging pain.

(*c*) **Symmetrical gangrene** is the outcome of repeated attacks of the above conditions. The parts about to become gangrenous exhibit the appearances met with in local asphyxia; small vesicles filled with sero-pus form at the finger tips; these burst, leaving minute sores. Temporary improvement may now occur, only to be followed by fresh ulceration. The finger ends usually taper and mummify, and the nails turn black. Sometimes the skin may slough and the nails fall off, the raw patches healing by granulation or remaining open.

FIG. 20.—Symmetrical gangrene of the great toes from a young woman who was the subject of Raynaud's disease. (Drawn by C. H. Freeman.)

In other cases the gangrene is more rapid, and there is no temporary improvement. Dry gangrene rapidly occurs and the dead part is separated from the living by a line of ulceration. The extent varies; all the fingers of both hands and part of the hands may die; sometimes the feet suffer simultaneously (Fig. 20).

As a rule there is no impairment of the general health unless constant pain has produced sleeplessness. Very often there is a distinct periodicity in the disease, the symptoms disappearing and recurring at intervals. It may extend over many months.

Diagnosis.—When gangrene is established, the diagnosis is

easy. Local syncope or asphyxia may be mistaken for chilblains. The periodicity, duration, and symmetrical distribution of the lesions, coupled with their occurrence at all times of the year, are the main diagnostic points. Simple senile gangrene is not likely to be mistaken for Raynaud's disease, the age of the patient alone being sufficient to prevent such a mistake.

Treatment.—Warmth and gentle friction should be applied during an attack of arterial spasm, and opiates may be necessary to relieve pain. Electricity has been found the most useful remedy among the many tried. The hand and one of the electrodes should be placed in a basin of hot salt solution and the other electrode higher up on the arm; the current should be as strong as the patient can bear it and should be frequently interrupted.

Iron, quinine, and good food should be given.

When gangrene has occurred, the sloughs should be left to separate, and if its extent requires amputation, this should be performed when the line of separation has fully formed.

GANGRENE FROM ERGOTISM

Ergot gangrene was at one time more or less common in France; it is now rarely met with. It occurred only among the very poorest peasants, whose food mainly consisted of rye bread, and who lived amid privations and hardships that were no doubt powerful predisposing causes. It was more common in men than women, and sometimes occurred in epidemics. The feet were more often affected than the hands, and the disease was sometimes bilateral and varied in extent. The gangrene was of the dry variety, closely resembling the senile form. Constitutional symptoms and even death were sometimes induced by the toxic properties of ergot.

Amputation, when the line of separation had formed, was the only treatment.

GANGRENE DUE TO DIATHETIC STATES—DIABETIC GANGRENE

Diabetic patients are peculiarly susceptible to low forms of inflammation terminating in sloughing or gangrene, and often resulting from the most trivial injury, such as the application of a blister.

Diabetes not only exhausts the patient and impairs general nutrition, but is frequently associated with arterial degeneration and

peripheral neuritis, and it has been already shown that these are in themselves powerful factors in the production of gangrene. It must be remembered that the presence of sugar in the urine is not necessarily diagnostic of diabetic gangrene. Transient glycosuria has often been noted, apparently as the outcome of any gangrenous process. The course and rapidity of the gangrenous process varies, and some authorities recognise three forms coinciding with the most prominent causative factors. Inflammatory gangrene of the moist variety runs an acute course characterised by extensive ulceration and sloughing, and may result from a slight injury or trivial inflammation.

When arterial degeneration is responsible for the necrosis, it assumes all the characters of senile gangrene, is accompanied by great pain, and runs a rather rapid course. The neuritic form, on the other hand, is painless, and progresses slowly, and may begin as a perforating ulcer.

Diabetic gangrene often begins as a bleb containing dark brown fluid, surrounded by an inflammatory zone; the neighbouring tissues participating in the inflammatory condition gradually perish, and the gangrene spreads.

Prognosis.—The prognosis is very grave, especially in rapidly-spreading cases, in the aged, and when the amount of sugar passed is large. Death may occur from exhaustion or diabetic coma.

Treatment.—As soon as the line of separation has formed, amputation must be resorted to, if the condition of the patient will admit. In the inflammatory, rapidly-spreading gangrene, a line of separation may never form, the patient quickly succumbing from exhaustion or acetonæmia. No doubt diabetics are not good patients for surgical operations, but recent work has shown that the dangers have been exaggerated. The strictest antiseptic precautions must be adopted, and the patient got well under treatment by opium and appropriate dietary. After removal of the gangrenous part the amount of sugar passed materially diminishes, although this improvement may not be permanent.

The point at which amputation should be performed varies with circumstances, and is a matter of importance. If there is evidence of marked arterial disease, amputation should be performed high up, as in cases of senile gangrene; but if the progress of the case is slow and unaccompanied by pain, neuritis being therefore the presumable cause, the limb may be removed just above the line of separation.

GANGRENE DUE TO MICRO-ORGANISMS

Certain micro-organisms are capable of inducing severe inflammation, resulting in sloughing or gangrene. These forms are fully discussed with the infective processes; they include boil, carbuncle, malignant pustule, cancrum oris, hospital gangrene, phagedæna, and acute emphysematous gangrene (see chap. vii. p. 102).

CHAPTER VI

Bacteriology in Relation to Surgery

The science of bacteriology may be said to date from the investigations of Pasteur (1857) into the causes of fermentation, a process long recognised as presenting a very close analogy to putrefaction and the infectious diseases. Pasteur showed that each kind of fermentation was dependent upon the presence of specific micro-organisms, and although some denied their influence, attributing their undoubted presence to accidental contamination, the germ-theory receives at present universal support.

Some organisms act by inducing, as the direct result of their own metabolic activity, chemical changes in the fermenting matter (organised ferments); in the case of others, the changes are brought about by means of unorganised ferments of a complex albuminoid nature, secreted by the organisms; such ferments excite the resulting chemical changes without themselves undergoing any alteration. Ptyalin and pepsin are examples of unorganised ferments in the body.

The clinical course of contagious and infectious diseases suggests the occurrence of processes analogous to those demonstrated as taking place in fermentation. The science of bacteriology has for its aims the discovery of organisms capable of exciting disease, the study of their life-history and mode of action, and of the conditions favourable or inimical to their growth and development.

There are many infectious diseases in which, at present, no definite organism has been found; but in other cases organisms have been isolated and proved by demonstration to be the *contagium vivum*. By analogy we may fairly assume that such causative factors will be proved to exist in all infectious processes.

Before we can be certain that any organism stands to a given disease in the relation of its cause, and not as a mere accident or association, certain conditions must be fulfilled (Koch's postulates)—

(1) The organism must be constantly present in the tissues or fluids of the diseased animal.
(2) It must be isolated, and pure cultivations of it obtained.
(3) The disease must be reproduced in animals by inoculation with the organisms from such cultivations.
(4) The same organism must be present in the tissues or fluids of the inoculated animals.

Micro-organisms belong to the lowest class of fungi, and are divided into three groups—

(1) The bacteria or schizomycetes.
(2) The yeasts or blastomycetes.
(3) The moulds or hyphomycetes.

The bacteria are by far the most important, since to this class belong, with few exceptions, those micro-organisms shown to be the causative elements in infectious diseases. The yeasts and moulds will not be considered here.

THE BACTERIA, SCHIZOMYCETES, OR FISSION-FUNGI

Structure and physical characters.—The bacteria are unicellular vegetable organisms devoid of chlorophyll; they belong to the lowest class of fungi, and are composed of delicate protoplasmic material—mycoprotein—enclosed by a protective sheath of the nature of cellulose. The protoplasm is often granular, and contains nuclear chromatine, but no definite nucleus has been demonstrated. In some cases the outer part of the investing membrane is gelatinous, the organism appearing to be surrounded with hyaline material; this material may bind together numerous microbes into zooglœa masses.

Most organisms are colourless, but some contain pigment, which is present in the protoplasm, in the investing membrane, or both, and which readily diffuses in nutrient media, wherein the organism may be placed for purposes of cultivation, and thus forms an aid to diagnosis.

Some organisms (*e.g.* bacillus tuberculosis) are motionless, others are motile. The latter variety possess flagella, developed usually at one or both ends, and sometimes laterally, as in the typhoid bacillus. The number of flagella varies; sometimes there is only one, but

there may be one or more at each end. These flagella, which are developed from the investing membrane and protoplasm, are, on account of their extreme tenuity, difficult of demonstration.

Some of the organisms are true parasites, *i.e.* they live in and at the expense of a host; others, known as saprophytes or carrion-fungi, live only on dead matter. The former are known as *pathogenic*, the latter as *non-pathogenic* organisms. Their effects and mutual relations will be more fully discussed later on.

FIG. 21.
Typhoid bacilli showing flagella.

Classification. — For practical purposes bacteria are divided according to their shape into micrococci, bacilli, and spirilla.

Micrococci are the smallest microbes known. They are round or oval in shape, and motionless. They germinate by fission only, never by spores, and this process of fission may give rise to a variation in grouping. Thus they may be found as single cocci, the individual organisms having no definite relation to each other; or when a coccus divides, the resulting cocci may remain close together, forming a pair (diplococcus, Fig. 34, p. 156); these again dividing in the same plane, chains or chaplets result (streptococcus, Fig. 10, p. 41). Organisms irregularly grouped together, as in zoogloea masses, are known as staphylococci (Fig. 9, p. 40). If an organism divides, and the resulting pair divide again in a plane at right angles to the first, a group of four is formed (tetrad); the process being repeated gives rise to a group of eight (sarcina), and so on. This grouping is not a mere matter of accident, but is characteristic of individual forms.

The micrococci which are known to possess pathogenic properties are those of suppuration, viz. staphylococcus pyogenes aureus and albus, streptococcus pyogenes, etc. (see p. 40). The M. ureæ excites putrefactive changes in urine; sarcina ventriculi is met with in the vomit from cases of pyloric obstruction.

Bacilli are straight, or but slightly curved, elongated, rod-shaped organisms. They differ considerably in size, and many of them, being armed with flagella, exhibit constant oscillatory or rotatory movements (Fig. 21). Some bacilli multiply by fission only, others by fission and by spores, the latter being very resistant to injurious influences.

Bacilli may form zooglœa masses, or, dividing transversely, may remain united by gelatinous material, and form long chains; in other cases they are not definitely grouped.

Bacilli are found in tubercle, syphilis, leprosy, rhinoscleroma, anthrax, glanders, diphtheria, influenza, typhoid, tetanus, noma, malignant œdema, plague, and septicæmia. The B. coli communis is constantly present in the intestine, and has pathogenic properties. The B. lactis aërogenes, bacillus pyocyaneus, and proteus vulgaris are occasionally met with.

Spirilla are motile organisms occurring either as long, closely-wound flexible spiral cells, or are shorter, more open and stiff. They germinate by spores.

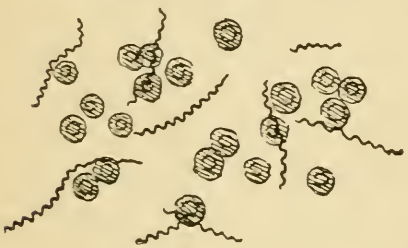

Fig. 22.—Spirilla and red blood cells.

Spirilla are met with in relapsing fever and cholera.

Life-history of micro-organisms.—Habitat.—Bacteria are abundantly present in nearly all things surrounding us; but, while thus universal in their distribution, they, like all other living things, grow better and multiply more abundantly in some media than in others, and under certain inimical conditions die, or fail to develop, although retaining the power to do so. Bacterial growth is specially prolific in organic matter with warm and moist surroundings. Air, especially if moist or dust-laden, and in the neighbourhood of defective sanitary arrangements or of infective centres, *e.g.* hospitals, is very rich in microbes. Dry air is nearly sterile, since all organisms require moisture. The surface soil is rich in bacteria, especially bacilli, but deeper down they decrease in number, and at about one metre none are found unless carried there by percolation of surface water. Deep spring water is almost sterile, but ordinary water is rich in bacteria, all of which may readily be killed by boiling. The surface of the skin and mucous cavities are favourable sites for bacteria, but in the case of the skin they cannot, in the absence of a wound, gain entrance to the body. Bacteria may possibly pass through the mucous membranes in small numbers, but are usually soon destroyed.

Conditions essential for life and growth.—Like other living things, bacteria require nutriment and favourable surroundings; but even if the conditions are not favourable to development, the organisms may remain living, though dormant, and capable of

growing and multiplying when the inimical conditions no longer exist.

Most bacteria of interest to the surgeon are pathogenic, *i.e.* they are true parasites and find in the body of the host pabulum suitable to their requirements. Parasitic microbes are *obligate* or *facultative;* the former cannot grow outside a host, the latter, although usually leading a non-parasitic existence, are capable of acting as true parasites.

The non-pathogenic bacteria are saprophytes or carrion-fungi, obligate or facultative; they can only subsist on dead matter, and if taken into the body, speedily die. In many cases, however, *e.g.* ill-drained wounds, and abscesses, stagnant urine, etc., there is in the body an ample supply of dead material in which non-pathogenic organisms thrive and produce their toxines, which, being absorbed, give rise to pathological phenomena.

Some organisms (*e.g.* B. anthracis) require free oxygen, others are killed by it, or at least sustain an arrest of development; the former are called *aërobic*, the latter *anaërobic*, and either may be obligate or facultative.

All organisms require water, carbon, hydrogen, nitrogen, oxygen, and salts, especially potassium and phosphates. Unlike plants, they cannot assimilate carbon-dioxide, since they do not contain chlorophyll. The necessary food elements are obtained by the splitting up of fats, albumens, carbohydrates, and water.

The influence of temperature is very marked, 30°-35° C. being that most suitable for growth. The limit of temperature for the growth of most organisms ranges from 5° to 50° C; nearly all die at boiling-point, and many at much lower temperatures. All organisms are much less injuriously affected by dry than by moist heat.

The spores are far more resistant to the effects of heat and other injurious influences than are the mature organisms, a fact of extreme importance in preventive medicine.

Conditions unfavourable to growth and development.—Temporary withdrawal or diminution in the supply of nutrient material, or unfavourable conditions as regards moisture, temperature, etc., necessarily impair the vital activity of micro-organisms, and, if of sufficiently long duration, prove fatal. Inimical conditions do not necessarily kill, for although the organisms may remain inactive for a long period (the spores very much longer), they will again grow and multiply under favouring circumstances. The influence of high and low temperatures has already been mentioned.

Desiccation is very harmful; nothing which is really dry can putrefy. Some organisms withstand desiccation better than others, and the spores of all are very resistant, as has already been stated.

Movement of the medium is unfavourable to nearly all organisms, and for many rest is essential. Most grow better in an alkaline or neutral medium. Bright sunlight is harmful, but probably has not much influence provided other conditions favour the organism. Certain substances, known as antiseptics and germicides, will kill micro-organisms or render their toxines innocuous (see chap. i. vol. ii.). Bacteria, as the result of their own activity, give rise, *inter alia*, to substances hostile to themselves; such substances are known as *anti-toxines*.

In common with other forms of life, bacteria are subject to the laws of struggle for existence and survival of the fittest; if any nutrient medium favourable to the growth of several kinds of microbes be inoculated with them, the weakest and least favoured will perish in the presence of the stronger and more fit. Thus a nutrient fluid containing sugar, into which numerous organisms have been introduced, will undergo lactic acid fermentation, the B. lactis being the most favoured; if, however, .5 per cent tartaric acid be added, torulæ gain ascendency and excite alcoholic fermentation.

While there is this obvious antagonism, there is also evidence to show that certain organisms act better in the presence of others. Thus ordinary putrefactive bacteria give rise to poisons which act deleteriously on the cells, and, if absorbed, on the body generally, hence they lessen the resisting powers and pave the way, so to speak, for the attacks of pathogenic organisms. Watson Cheyne has pointed out that tubercular joints with septic sinuses are much more often complicated by general tuberculosis than are similar joints without septic sinuses; and it is further known that staphylococcus pyogenes aureus and albus act much more powerfully when combined (see p. 40).

Products of bacterial action.—The chemical products of bacterial action differ in different organisms, and in the same organism under varying composition of the nutrient medium. Some of them are harmless, and therefore of no clinical importance; others are, in varying degrees, poisonous (*toxines*); while a third group are antidotal and protective (*anti-toxines*).

As yet the precise nature of these products is almost unknown, although unformed ferments, toxic alkaloids, and albumoses have been isolated in many instances. It would appear that toxines

may be secreted by the organism itself, or may arise from chemical changes occurring in the media as the result of the vital activity of the organism. Such toxines are very soluble and, being absorbed into the blood, produce definite chemical effects, sometimes on the body as a whole, at others showing a special predilection for certain tissues (*e.g.* diphtheritic paralysis, in which the diphtheritic poison specially affects the nerves). In some cases the toxines are bound up with the microbic protoplasm from which they are not readily freed, and hence such organisms possess toxic properties even when dead.

Reproduction by fission has already been alluded to above (p. 89).

Some bacilli germinate by spores as well as by fission. When a spore-bearing microbe has attained its full development, it becomes granular, and a series of highly refracting bodies—the spores—appear in its interior, where they remain until freed by degenerative changes which lead to a splitting up and disintegration of the parent organism. These spores are composed of protoplasm and an investing cellulose sheath of considerable density (whence probably their great powers of resistance to injurious agents); they develop into the mature organism, and the process is repeated indefinitely.

FIG. 23.—Anthrax bacilli and spores.

Mutability of species.—Some pathologists have maintained that all micro-organisms belong to one species, the various recognised forms being merely stages in development. This opinion is, however, held by very few, and is opposed to the weight of evidence. Clinically we never see anything consistent with mutability of species; on the contrary, we know that certain parasites invariably produce identical pathological phenomena, no matter through how many generations they may have been cultivated. In such cultivations, moreover, the organisms always behave in the same way, exhibiting the same affinities and dislikes.

Cultivation through many generations has never shown that a micrococcus can become a bacillus or spirillum. In the face of these important negative facts, and in the absence of any sound confirmatory observations, we may safely assume that unity of species is highly improbable.

Method of invasion.—Non-pathogenic fungi, being incapable of existing in living tissues, are powerless for evil and soon perish when taken into the body by means of food or air. In ill-drained abscesses and wounds the discharges consist of dead material, and afford fitting pabulum for these microbes which are thus enabled to thrive and multiply; if their soluble toxines are absorbed by the lymphatics, general septic poisoning is produced (Septic Intoxication). The organisms are introduced by dirty instruments, sponges, fingers, and the like.

Pathogenic fungi, which are capable of living in the fluids and tissues of the host, may be similarly introduced, or may enter by the alimentary or respiratory tracts, especially if there be any inflammation or lesion of the mucous membranes. Unhealthy wounds are more suitable for bacterial invasion than are healthy ones, since the vitality and natural resistance of the tissues is diminished. Again, recent wounds are more liable to pathogenic infection, and to absorption of the toxines of non-pathogenic organisms than are granulating wounds, since granulations do not contain lymphatics (the paths by which absorption and dissemination occur), and thus offer a barrier—though not an insuperable one—to infection.

Mode of action of organisms.—**Non-pathogenic microbes** act by means of the toxic properties of the products of their activity; they remain strictly localised to the dead matter in which they live, and the general symptoms are directly proportional to the amount of toxine absorbed (Septic Intoxication).

Pathogenic organisms, on the other hand, spread from the seat of inoculation, multiply in the tissues or fluids, and induce symptoms out of all proportion to the amount of the original dose.

It does not, however, necessarily follow that inoculation is followed by infection, since the dose may not be sufficiently large, or the animal may prove refractory or immune. Some pathogenic organisms may remain strictly localised to the point of inoculation (B. tetani and diphtheriæ); some spread by continuity of tissue only, others by the lymphatics or by the blood, either entering the circulation directly through the vessels, or indirectly through the thoracic duct. Thus, wound diphtheria is a purely local process showing no tendency to spread; hospital and spreading traumatic gangrene spread by continuity of tissue, while soft chancre and cellulitis spread by the lymphatics, and acute septic infection through the blood-stream.

Organisms gradually invading the body by local infection give rise to the *local infective processes* and cause general symptoms by absorption of their toxines; those spreading by the blood-stream induce *general infective processes*, and the organisms, sometimes carried to distant parts, excite inflammatory changes and secondary centres, each of which is a focus for further infection.

The effect of toxines on the tissue cells is to lower their vitality and lessen their powers of resistance, so laying them open to attacks from without; thus we see a point of connection between non-pathogenic and pathogenic fungi, the toxines of the former lessening the resistance of the tissues, and thereby producing or increasing a predisposition to infective processes. In many diseases (*e.g.* typhoid, diphtheria, tetanus) the gravity of the symptoms does not depend on the local manifest lesions, but upon the absorption of toxines or toxine-forming materials.

Proneness to infection — predisposition of the host. — By predisposition we mean that vulnerability of the body or tissues which makes it or them peculiarly liable to become the seat of infection and morbid processes, and which, moreover, influences to some extent the course and severity of such disease.

Animals which are proof against infection by any special organism are said to be *immune* to it, while those which are inoculated with difficulty are spoken of as *refractory*. Although we know that many conditions predispose an animal to disease, or render it immune, we do not know how these conditions act. Predisposition is hereditary or acquired. Hereditary predisposition may be accentuated by any condition which has a prejudicial influence on the health, and may be increased or diminished by interbreeding. Again, heredity may protect a whole species, or only certain individual members. Thus we know that the tubercle bacillus is especially fatal to guinea-pigs, but that rats are refractory; house-mice are speedily killed by a septicæmic virus to which field-mice are immune; anthrax easily affects mice but not rats; lastly, syphilis is probably peculiar to man (see p. 169). With regard to individual susceptibility it is a matter of common experience that during epidemics some persons are affected, whilst others, equally exposed to infection, and apparently not more robust, escape. It is well known that erysipelas is very prone to repeatedly attack some persons in presence of the most trivial wound.

Age is a potent factor; thus diphtheria, scarlet fever, measles, and acute necrosis especially attack the young, the last being con-

fined to children; anthrax kills young dogs easily, whilst old ones are refractory.

With regard to acquired predisposition, many factors favour the development of infective processes. Wounds and mechanical injury predispose in two ways—(1) by lowering the vitality of the tissues, and (2) in some cases where extravasation occurs (*e.g.* bruising), organisms that are present in the blood, but incapable of growing in it, find their way into the damaged tissues, where development can occur. The seat of a wound (in other words, the point of inoculation) may be favourable or hostile to the growth and spread of a particular organism; thus the bacillus of malignant œdema develops only in connective tissue, and if present in the blood is incapable of doing harm.

Diseases of the *primæ viæ*, especially of the kidneys, chronic alcoholic poisoning, and faulty hygienic surroundings certainly act as strong predisposing factors. No doubt in all these cases the retention of nitrogenous waste products in the body, by affording ample pabulum for the organisms, is a most important element in predisposition. If the kidneys, skin, etc., do not act properly, effete matters are not excreted in sufficient amount, and remain stored in the body. Diseases of the heart or lungs, bad air, and overcrowding, limit the amount of oxygen; oxidation does not therefore take place to the full extent, and nitrogenous material is present in excess. Chronic alcoholism, by diminishing oxidation, leads to a like result.

A certain dose of poison is necessary before any effect is produced, but this dose varies according to the degree of predisposition or immunity; the greater the predisposition, the smaller is the dose needful for inducing disease.

Micro-organisms of the same species have not always the same degree of virulence; it may be increased or attenuated, either by circumstances naturally occurring or artificially induced; thus, cowpox is in all probability only an attenuated form of smallpox. Attenuation of a virus naturally diminishes its power for evil, and a larger dose is necessary to produce results.

Immunity.—An animal is said to be immune to an infectious disease when inoculation fails to produce it.

Immunity, like predisposition, is natural or acquired by inheritance and natural selection among those subjected to the influence of any given pathogenic organism, the fittest and least susceptible surviving and transmitting their powers of resistance to their offspring. Yet this does not explain in the least what the nature of

the physiological difference in the cells or fluids of susceptible and immune animals may be; neither are the changes understood by which such cells or fluids in susceptible animals become immune after being once attacked. One attack of a general infective disease (*e.g.* smallpox) confers immunity, though probably only for a certain length of time; such immunity is not, in the majority of cases, acquired by attacks of the local infective processes.

Immunity may be conferred on an animal artificially, but does not endure for ever; thus, in vaccination, immunity to smallpox only extends over a limited number of years unless the operation be repeated. Such artificial immunity may be produced by inoculation with the attenuated virus, or with small doses of its toxines; the latter method has this great advantage over the former, that as no living organisms are introduced, there can be no increase of the poison in the body.

The products of the virus, used in small doses, probably prepare the cells to tolerate larger ones, just as we see toleration established in opium and arsenic eaters; or it may be that the virus so alters the chemical composition of the media that the organisms no longer find the pabulum suitable to their requirements.

The blood serum of an animal rendered immune *artificially*, if injected into a susceptible animal, renders it immune; or, if it be already attacked by the disease, serves to check its course and severity, and to bring about recovery. This fact is made use of in the anti-toxine treatment of diphtheria and other conditions. What the substance or substances are upon which this protective property of the serum depends we do not know; they are provisionally called anti-toxines or defensive proteids. It must not be forgotten that the blood serum of a healthy animal possesses germicidal properties, but blood serum as such is not present in the living body. It has not been shown that the blood serum of an animal *naturally* immune to any disease confers immunity on susceptible animals.

It is highly probable that the method of production of immunity is not always the same, and one would naturally expect that, as the methods of invasion and growth of different organisms vary, so would also the means of protection.

At the present time there are two leading views as to the nature of immunity; some hold that the changes are vital (phagocytosis), others that they are chemical.

Phagocytosis.—Metchnikoff and his followers maintain that certain of the leucocytes and the endothelial cells of the capillaries

and lymphatics are either naturally, or may be artificially rendered capable of destroying poisonous organisms, and thus protect the host against invasion. In susceptible animals these cells, termed *phagocytes*, possess this power in a limited degree or not at all. The theory of phagocytosis suggests that on the introduction of any organism or noxious matter into the body, a war for supremacy is waged between it and the phagocytes, the result to the host depending on the issue of the combat. If the phagocytes are very active, and the organisms possess only slight powers of resistance, the latter are speedily destroyed, and their characteristic pathological effects are not produced—in other words, the host

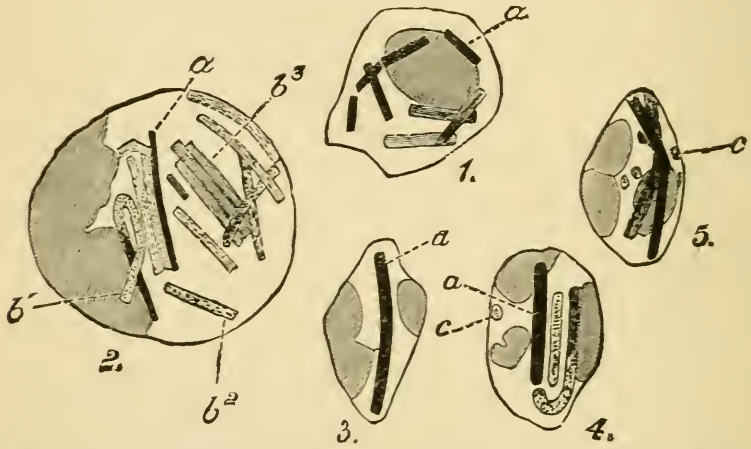

FIG. 24.—Anthrax of pigeon (an animal only slightly susceptible to the disease) to show the stages of destruction of bacilli by phagocytes. 1, macrophage from exudation from the eye of refractory bird; 2, macrophage from muscle of region of inoculation of bird that succumbed; 3, 4, 5, microphages from the eye twenty-seven hours after inoculation; *a*, *a*, unaltered bacilli; b^1, b^2, b^3, bacilli becoming more and more degenerated and indistinct; *c*, *c*, débris of bacilli. (Allbutt's *System of Medicine*, after Metchnikoff.)

proves immune; if the disproportion in strength of the combatants is not so marked, the disease is contracted in a mild or severe form; lastly, if the organisms are much stronger than the phagocytes, they invade the host and bring about a fatal result.

Phagocytic cells are chiefly derived from the leucocytes, of which the blood contains four forms:—

(1) Lymphocytes—cells with a single round nucleus and a little protoplasm.

(2) Mononuclear leucocytes or macrophages, similar to lymphocytes, but larger.

(3) Eösinophile leucocytes, with a large lobed nucleus and granular protoplasm. They are formed in the bone-

marrow, and derive their name from the fact that they stain only with acid aniline dyes, such as eösin.

(4) Polynuclear leucocytes or microphages. The nucleus is lobed or multiple.

Of these cells, the macrophages and microphages alone act as phagocytes.

Vascular and lymphatic endothelial cells are capable of acting as "fixed" phagocytes, but are also sometimes found free in the vessels. Phagocytes are sensitive cells, and are attracted or repelled by certain substances or organisms; attraction is spoken of as positive, repulsion as negative *chemiotaxis*, and indifference on the part of the leucocyte is called neutral chemiotaxis. The chemiotactic state of the cells as regards any organism is a matter of great moment; positive chemiotaxis determines a migration of leucocytes to the damaged or invaded spot, where they then endeavour to ingest and destroy the invaders; negative chemiotaxis, by repelling the leucocytes, enables the organisms to invade the body.

In support of the theory of phagocytosis, the following observations are of importance. It is well known that amœbæ ingest and live upon bacteria and low forms of fungi. In higher organisms, with differentiation of structure, the same process occurs; thus, in sponges, the mesoderm cells act as phagocytes; and Woodhead has noticed that in the growing cod's ova all the cells take up the yolk masses in the early stages, but that as development proceeds this function is confined to the mesoderm cells alone. Phagocytosis also plays an important part in the evolution of certain larvæ; thus the tadpole's tail is removed by phagocytic action. We know, moreover, that catgut ligatures and effete material are removed by leucocytes; yet in these cases it must be observed that the ingesta are not living, and have no powers of resistance other than their physical properties give them.

When anthrax bacilli are injected into the blood-stream of a frog, they are ingested and destroyed by the leucocytes, and Metchnikoff has shown that when daphnia (water-flea) is attacked by monospera, the leucocytes show marked activity (*positive chemiotaxis*), crowding round and destroying the parasite; but if daphnia is invaded by another parasite (saprolegnia), the leucocytes are peculiarly impassive (*negative chemiotaxis*), the parasite thus gains ground and kills the host. In fatal cases of erysipelas in man, the organisms are not taken up by the leucocytes, but in more favourable cases they are, very few being then found in the tissues. Animals immune to any organism will, if the spleen be previously

removed, rapidly succumb to inoculation; this fact certainly tends to support the theory, since the spleen is the factory for leucocytes, but unfortunately its value is discounted by our ignorance of the functions of the organ.

While the fact of phagocytosis is acknowledged by all, the part claimed for it in the protection of the host is disputed. Metchnikoff's opponents do not deny that the phagocytes assemble round the areas of bacterial invasion; yet they contend that this is not for purposes of aggression, but merely because they find in the dead and dying tissues and organisms a pabulum suitable to their needs; and that when living organisms are met with in the cells, they are the aggressors, and are actually killing the leucocytes. Doubtless many of the leucocytes do perish in the struggle for supremacy. It must be remembered that many organisms are non-motile, and in the case of these, at least, it is impossible that they could have penetrated the leucocytes; they must therefore have been taken up by them.

The chemical or humoralist theory asserts that organisms are killed, not by cells, but by the fluids of the blood. It has already been stated that the serum of animals rendered artificially immune confers, when injected, immunity on susceptible animals. Blood serum may, it is stated, exert a protective influence in two ways—(1) by a definite bactericidal action on the organisms themselves, and (2) by antagonising the toxines of bacterial action, and rendering them harmless. The two processes may be combined.

The defensive action of serum is said to be due to the presence of nuclein, derived by the disintegration of leucocytes (phagocytes). This, if true, is in part at least an acknowledgment that leucocytes have themselves the power of killing organisms, since it is only reasonable to suppose that if leucocytes contain a substance possessing germicidal properties, they can themselves be germicidal. The only alternative to this proposition lies in supposing that, as in the case of the fibrin-ferment, the toxic properties of nuclein are only developed when it is freed from the cells and mixed with the fluid parts of the blood. Against this, Woodhead has shown that animals in which the leucocytes have been extensively destroyed by quinine, are more susceptible to anthrax than they were before, an observation materially strengthening the phagocytic view. To test the bactericidal action of the fluids of the blood, Schultze introduced into the veins and lymph sacs of animals and frogs small boxes made of rose-pith, and filled with the spores of pathogenic organisms. The filtering action of the pith kept out the leucocytes,

but allowed fluids to pass into the boxes. Schultze found that so far from being killed, the spores developed and multiplied.

Future experiments will no doubt throw more light on this question of immunity, and it seems likely that the two theories may be harmonised. Some authorities have endeavoured to reconcile the two views. They point out that the fluids of the body must be regarded as the expression of the vitality of the cells, that any properties these fluids possess are due to the activity of the cells, and that any cell capable of conferring anti-toxic properties on a fluid must surely itself be anti-toxic.

It is possible that the organisms themselves are destroyed by the phagocytes, while their toxines are neutralised and rendered harmless by the fluids, the anti-toxic properties of which may be increased by—or may even depend upon—the breaking down of those phagocytes which perish in the combat.[1]

[1] For further information, see *The Comparative Pathology of Inflammation*, by Metchnikoff.

CHAPTER VII

Surgical Septic and Infective Diseases

Definition and classification.—The word septic is often used as a generic term applied to diseases, the symptoms of which are chiefly dependent upon the action of poisons due to the presence of micro-organisms. This generic use of the word too frequently gives rise to confusion, especially in the mind of those who are commencing the study of surgery, and it should be used in a very much more restricted or qualified sense.

Septicæmia and pyæmia are terms similarly employed in a very loose way; although sanctioned by long usage it would be much better if the former at least were abandoned, including as it does pathological processes of very different aspect, though at the same time they have this in common—they are all due to poisoning. It is proposed here to adopt that nomenclature and classification which appears to be the best and the most likely to convey exact ideas as to the nature of the diseases to be considered in this and succeeding chapters.

Simple septic diseases are those which are due to the absorption of poisons generated by ordinary putrefactive decomposition. The organisms producing these poisons are non-pathogenic, and hence are incapable of living in the body unless there is some dead material, *e.g.* pus, blood-clot, or urine present which serves as a suitable pabulum. The clinical symptoms induced are directly proportional to the dose of poison absorbed. The conditions belonging to this group are—

(1) Acute septic intoxication.
(2) Chronic septic intoxication or hectic.
(3) The constitutional effects of poisoning by decomposing articles of dietary, or in some cases (*e.g.* fish-poisoning) by fresh food.

Infective diseases are dependent on the absorption of poisons produced by pathogenic organisms which are capable of multiplying in the living body quite irrespective of the presence of dead material, and hence of inducing symptoms out of all proportion to the amount of the original dose. These diseases may be local or general; but it must be understood that many which are here classed as local may terminate fatally by general infection. Such a termination may be due to mixed infection, or may depend upon the fact that organisms usually remaining local, may under favourable conditions be absorbed into the blood and cause general infection.

A. *Local infective diseases* are excited by microbes which spread locally only and not by means of the blood-stream, although their toxines can be and are absorbed and give rise to constitutional symptoms.

The following diseases fall under this category:—

(1) Boil, carbuncle, and facial carbuncle.
(2) Malignant pustule (external anthrax).[1]
(3) Cancrum oris, noma vulvæ.
(4) Hospital gangrene.
(5) Sloughing phagedæna.
(6) Wound-diphtheria.
(7) Cutaneous erysipelas.[2]
(8) Cellulo-cutaneous erysipelas.
(9) Cellulitis.
(10) Emphysematous gangrene.
(11) Rabies.[3]
(12) Tetanus.
(13) Actinomycosis.
(14) Madura foot.
(15) Tubercle.[1]
(16) Gonorrhœa.[1]
(17) Chancroid.

B. *General infective diseases* are those due to the presence of pathogenic organisms which gain entry into the blood current,

[1] Malignant pustule, tubercle, and gonorrhœa may become general diseases. The first is really the effect produced by the local inoculation of anthrax, but early and radical treatment may prevent general infection by the blood-stream. Gonorrhœa is essentially a local process, but in some cases of gonorrhœal rheumatism the gonococcus has been found in the synovial effusion.

[2] Opinions are divided as to whether erysipelas should be regarded as a local or general infective process (see p. 123).

[3] As the organism of rabies has not yet been discovered, the disease is provisionally placed here.

either directly through the veins or indirectly through the lymph channels, and thus produce multiple foci of disease.

The diseases included under this heading are—

(1) Septic infection.
(2) Septic infection with secondary centres of suppuration.
(3) Glanders.
(4) Syphilis.

The relation between septic and infective processes.—The occurrence of mixed infection has been already alluded to at p. 95. There is no necessary connection between a septic and an infective process, but in practice we find that when a wound is the seat of an infective process, putrefactive decomposition and simple septic absorption are also present. It must be borne in mind that this does not necessarily imply the association of different forms of organisms, because many pathogenic microbes are also facultative saprophytes, being capable of causing an infective and a simple septic process.

It is of paramount importance in preventive treatment to thoroughly appreciate the fact that a septic condition of a wound is the most favourable for the development of an infective organism. Putrefactive decomposition of discharges predisposes to infection in two ways:—

(1) By lowering the resisting powers of the tissues by the local and general effects of the poisons absorbed.
(2) By setting up inflammation and consequently providing discharge which serves as an excellent medium for the development of pathogenic organisms, many of which are, as already stated, facultative saprophytes.

If infection is the primary process, the inflammation at the seat of inoculation is accompanied by copious exudation, and thus dead material is provided which serves as a suitable culture medium for any saprophytic organisms which may be present. Doubtless in all the infective processes the general disturbance is partly due to the toxic effects of the poisons formed in the process, and partly to ordinary septic intoxication, and hence it is easy to see why the symptoms may show improvement when the wound is thoroughly drained and cleaned so that putrefaction is prevented or the absorption of its products hindered.

Causes favouring the development of septic and infective processes.—The mere presence of organisms is not in itself sufficient to excite the pathological changes consequent on their action; there must be certain favouring conditions of their environ-

ment. This subject has already been discussed at p. 90, and here it will only be necessary to briefly review the factors which are of the greatest practical importance to surgeons. It must be remembered that these predisposing conditions do not always bear the same relative proportion in etiology, for we know that organisms are sometimes more virulent than at others, and that certain persons, animals, or tissues are naturally predisposed, refractory, or immune to their influence, see p. 95.

The predisposing cause, *par excellence*, is faulty hygiene, especially overcrowding of surgical patients. Nothing is more certain than this; the improvements in modern sanitation and the introduction of antiseptics have virtually abolished many, and rendered comparatively rare nearly all of the surgical infective diseases.

A wound, although it may be of minute size, is always present; accidental wounds are more often the seats of these processes than are surgical ones, since the latter are from the first carefully protected from contamination.

Punctured, lacerated, and ill-drained wounds are for reasons already given (see p. 94) the worst. When granulations (which are destitute of lymphatics) have sprung up, they offer a barrier to the absorption of poisons and the invasion of the tissues by pathogenic fungi, and hence recent wounds are most liable to infection.

The employment of antiseptics, and above all, the complete cleansing of accidental wounds, is an essential in the sanitation of injuries, especially in hospitals and crowded cities. Leaving out of the question individual inherent predisposition, all causes (*e.g.* alcohol, and diseases of the *primæ viæ*) which lower the general health also favour the occurrence of infective processes, the weakened tissues being unable to cope with the invading organism.

General preventive treatment.—A knowledge of the etiological factors in septic and infective diseases is a guide to the general principles of preventive treatment. Free drainage of wounds, the strict employment of antiseptics, and hygienic surroundings are essential. Before an operation is performed the patient's general health should be brought as far as possible up to a normal standard.

SIMPLE SEPTIC DISEASES

The organisms capable of inducing putrefaction of organic matter are known as saprophytes or carrion-fungi. Those most

usually met with are the bacterium lineola, micrococcus prodigiosus, bacillus coli communis, bacillus lactis aërogenes, and proteus vulgaris, but many pathogenic fungi are facultative saprophytes. The products of decomposition vary with the organism inducing it, with the nature of the decomposing material, and with the conditions under which it occurs. Some are harmless, others poisonous, the poisons varying in their virulence. These poisons are all known as ptomaines or cadaveric alkaloids.

Sepsin, cadaverine, and putrescine are common; muscarine, collodine, parvoline, and others are found, especially in decomposing fish. Simple septic diseases only require that the poisonous products should be absorbed into the system. In recent wounds the serum and blood-clot are dead material, ready to putrefy in the presence of the ferment; pus, urine, and other discharges are similarly capable of putrefaction.

But even should putrefaction occur, it is by no means certain that absorption of the ptomaines will follow. If, as in the case of an open or well-drained wound, the putrefying discharges find easy exit, absorption is not likely to occur; but if there is any tension, the lymphatics and capillaries readily take up the ptomaines. In the case of granulating wounds, the granulations, being devoid of lymphatics, offer a bar to absorption; but even in these it may occur if tension be high, the poisons then percolating through the granulations to the lymphatics beyond.

Not only may absorption take place through wounded surfaces, but should the ptomaines be taken with the food, they will be absorbed by the gastro-intestinal mucous membrane (as in poisoning by sausages, fish, or by putrid pus from the lungs, mouth, etc.), and produce serious or even fatal consequences. This fact is of extreme importance from a medico-legal point of view.[1]

Ptomaines are excreted by the urine and fæces.

ACUTE SEPTIC INTOXICATION—ACUTE SAPRÆMIA

Symptoms.—The symptoms usually make their appearance during the second day after the injury, and are ushered in by chills or a distinct rigor, with a rise of temperature varying from 101°-104° F., which remains continuous, or presents slight intermissions. Vomiting, nausea, anorexia, thirst, and constipation are usually present, but in severe cases there is profuse watery diarrhœa with

[1] For a full account of septic absorption through the alimentary tract, the reader is referred to works on medicine.

perhaps bloody mucus. The tongue is furred, the breath foul, the skin hot and dry, the urine scanty, rich in lithates, and sometimes albuminous. The pulse is rapid and perhaps irregular, and headache is usually severe.

If the dose has been large, collapse and nervous prostration are prominent features; the tongue becomes dry and covered with sordes, and there is diarrhœa and profuse sweating. The headache passes off as delirium comes on, and this deepens to coma before death.

The amount of the dose and the rapidity of its absorption cause much variety in the effects and symptoms. The onset may be tumultuous and death rapidly ensue, but such cases are hardly ever seen in surgical practice; they may, however, occur in poisoning by putrid food, fish, etc.

In other cases the symptoms are but little marked, and come on more gradually, so that with prompt treatment the patient runs but little danger. All grades of severity may be met with.

Diagnosis.—The diagnosis from septic infection is at first impossible; it must be made on the course which the case runs and the effects produced by the removal of all putrid matter. From simple aseptic traumatic fever, sapræmia may be distinguished by the greater severity and more lasting nature of its symptoms, and by evidences of decomposition which are wanting in the former state (see chap. ii. vol. ii.).

Prognosis.—Until it is clearly ascertained that no infective process complicates the condition, the prognosis should be guarded. Acute sapræmia in surgical practice is not usually a serious affection, provided means be taken to prevent any further absorption. The symptoms usually disappear in from two to seven days. Owing to extensive destruction of the red blood cells the patient is rendered more or less anæmic.

Post-mortem appearances.—Decomposition sets in rapidly and the abdomen and tissues soon become distended with gases. There is marked *post-mortem* staining from decomposition of the red cells, brought about by the action of the poison.

The chief noticeable characters about the viscera are acute congestion with cloudy swelling, and occasionally petechial hæmorrhages which are especially noticeable on serous membranes, the secretion from which is slightly increased. The spleen is enlarged, soft, and may be diffluent.

Treatment must be directed (1) to the prevention of further absorption; (2) to combating the effects of the poison already in the system; (3) to improving the patient's health.

Further absorption is prevented by thoroughly cleansing the wound, by rendering it aseptic, and by providing free drainage, which may necessitate its enlargement or else counter-openings. Any inflammatory state of the wound must be treated by such means as seem suited to the case, and which have already been discussed in the chapter on Inflammation.

These measures alone are usually followed by a rapid fall of temperature and marked general improvement; should this not be the case, the prognosis becomes grave, as there is probably some associated infective condition.

So far we do not possess any direct antidote to the cadaveric alkaloids. They are excreted with the urine and fæces, and therefore the bowels should be opened and kept acting regularly, but violent purging must be avoided, as it adds to the prostration. The action of the skin and kidneys must be promoted by diaphoretics, diuretics, and demulcent drinks. The general rules of treatment in fever cases must be followed, and stimulants given in quantities regulated by the state of the pulse (see p. 31).

Sapræmia leaves the patient weakened and anæmic, and as soon as his digestive organs permit he should be supplied with a generous meat diet. Iron, quinine, and the mineral acids with small doses of strychnia are the best tonics. The patient should be sent to the country as soon as possible.

CHRONIC SEPTIC INTOXICATION—HECTIC

If repeated small doses of the cadaveric alkaloids be absorbed, the patient falls into a condition known as hectic or chronic septic intoxication. The difference between this condition and that just described may be familiarly likened to that between a man who occasionally and one who habitually takes an excess of alcohol. Hectic is especially likely to occur in connection with chronic suppuration of bones and joints, and especially large spinal abscesses and pulmonary cavities which have become putrid and are imperfectly drained; it cannot, of course, occur so long as the abscess remains unopened, as until then the saprophytes cannot gain an entry.

Symptoms.—Hectic is marked by general decline of health, accompanied by a nocturnal rise of temperature of one or two degrees with morning remissions. Anæmia from destruction of the red cells by the ptomaines is very marked; the patient becomes pale and waxy-looking, and muscular weakness is pronounced.

Contrasting with the general pallor is the well-known hectic flush on the cheeks. The tongue is tremulous (evidence of nervous weakness), pale, flabby, and teeth-indented. The appetite is indifferent and capricious, and emaciation keeps pace with the decline of the general health and vigour. As the case progresses, diarrhœa and profuse sweating — the latter occurring in the early hours of the morning—add to the patient's weakness. The urine is scanty, high-coloured, and rich in lithates. The pulse is frequent, and the heart fails in proportion to the general weakness. Delirium does not usually occur.

Prognosis.—Hectic is necessarily fatal unless the suppurative process can be stopped or means adopted to provide free drainage and prevent decomposition and absorption.

Treatment is conducted on the same lines as in acute sapræmia.

If the suppurative process cannot be checked by less radical means, amputation should be performed when the disease affects one of the limbs.

LOCAL INFECTIVE DISEASES

The virulence of the organisms inducing these processes varies within the widest limits (compare wound-diphtheria and hospital gangrene). Some of them remain quite localised to the seat of inoculation (*e.g.* B. tetani, B. diphtheriæ); others spread by local invasion only, *i.e.* by direct continuity of tissue; others again, *e.g.* cellulitis, spread by continuity of tissue, and also by the lymph paths, as evidenced by the acute lymphangitis and lymphadenitis they occasion. In many of the local infective processes the local results are of small importance, the danger lying in the absorption of the toxines (as in tetanus); while in others the local state may prove of a very serious nature (cellulitis, diphtheria, etc.)

The toxines are excreted with the fæces and urine.

FURUNCLE OR BOIL—CARBUNCLE

Boil and carbuncle differ in the intensity and extent of the destructive process rather than in their essential nature or cause. Boils often appear in crops; carbuncle is single, and may be compared to a closely-set group of boils. A boil remains localised, but a carbuncle may spread widely in the subcutaneous tissue. Moreover, the former is of no serious importance, while the latter may be attended with the gravest results.

Causes.—Boils chiefly attack young patients, whereas carbuncle is more common in adults.

General debility, diabetes, bad food, with an excess of nitrogenous matter, and bad hygienic conditions, are favouring causes. Friction is frequently responsible for the occurrence of a boil or carbuncle; both are common about the neck, especially posteriorly, over the shoulders, and on the buttocks, all of which are common seats of friction. Pyogenic organisms are met with in both conditions, the staphylococcus pyogenes aureus being the most constant and virulent (see p. 40).

Morbid anatomy.—The organisms gain entrance through a hair-follicle or a sebaceous or sweat-gland, and excite acute inflammation of the true skin, which extends to the subcutaneous connective tissue, but rarely involves the deep fascia. The inflammation remains localised, and the tension of the exudate leads to rapid sloughing.

In the case of a boil the slough is surrounded by pus, lying, in fact, in the middle of an acute abscess which bursts by one central opening, and, after separation of the slough, rapidly heals.

The slough formed in carbuncle is similar to that of a boil; it is ashen-gray in colour, and composed of dead connective tissue, infiltrated with leucocytes and coagulated lymph containing micro-organisms. Numerous small pustules form on the surface, and pus escapes through several openings in the thin and undermined skin. Many of these coalesce in the centre by destruction of the intervening skin, and the slough is exposed at the bottom of a ragged opening.

On separation of the slough, healing usually occurs without trouble. Infective thrombosis, followed by general infection, may result.

Signs and symptoms.—The signs of boil are too well known to require description. A carbuncle is usually oval or circular in shape, and may attain a large size; it is raised above the surrounding parts, and has an indurated, usually circumscribed base. The surface of the skin is livid and congested, and soon necroses.

A carbuncle sometimes shows a marked tendency to spread, often in one direction only.

The pain occasioned by boil or carbuncle is generally severe, especially in naturally tense regions, or where the skin is tough, *e.g.* the neck. Constitutional symptoms are not usually present in cases of boils; but in carbuncle there is more or less fever, with asthenic symptoms.

Prognosis of carbuncle.—In some cases the disease may

prove very serious, and the patient dies of exhaustion or general blood-poisoning. In diabetics, coma may supervene and prove fatal. Evidence of phlebitis is a grave sign, since infective emboli may be carried to different parts, and set up secondary areas of suppuration. Carbuncles approaching the scalp are serious, as the veins of the skull may be affected.

Renal disease, or any serious constitutional mischief, naturally increases the gravity of the case.

Carbuncle may last two or three weeks, or as many months.

Treatment of boil.—When a boil is threatened it may frequently be prevented by removing all sources of irritation and protecting the inflamed part. If a hair is present in the centre, it should be removed. Moist heat in the form of fomentations, or bathing with hot water, may cut short the process and prevent suppuration, or will hasten it if imminent. Should there be much pain, a boil may be advantageously incised; otherwise it may be left to burst, and the separation of the slough and the healing process be hastened by hot boracic fomentations. Repeated boils usually indicate weak health, which must be treated appropriately to the conditions causing it. Sulphide of calcium is said, perhaps without sufficient reason, to be useful.

Treatment of carbuncle.—Any constitutional disorder, *e.g.* diabetes, must receive its proper treatment. Alcoholic and diffusible stimulants, such as ammonia and ether, with quinine and bark, are required in view of the asthenic state of the patient. Opium, in gradually increasing doses, is a most valuable remedy, especially in diabetes; but it must be very cautiously given in cases of renal disease.

The action of the bowels and kidneys must be promoted. The food must be light and easily digestible, and if there is much fever, slops only should be given; but as the general state of the patient improves, a more liberal diet may be allowed with advantage. With regard to *local treatment*, opinion still differs as to the employment of incisions; they are undoubtedly beneficial if tension and pain are great. The incision should be made through the indurated mass, hæmorrhage being arrested by pressure. Some surgeons recommend its being followed by removal of the slough with the sharp-spoon; but this is rarely advisable, being a rather more severe procedure than the patient's condition warrants. The injection of carbolic acid into the sloughing mass has met with some success, and is worthy of trial, especially in cases where more active treatment is inadmissible or dangerous.

All antiseptic precautions must be adopted, and hot boracic fomentations should be applied until granulation is established.

FACIAL CARBUNCLE

So-called facial carbuncle is considered by some as a separate disease; but it is probably only an ordinary carbuncle running a more severe and fatal course, on account of its position in highly vascular structures. It occurs in young patients, usually between twenty and thirty years of age. Facial carbuncle is usually seen on the upper lip. It begins as a small itching pimple, and the lip becomes brawny, livid, and painful. The swelling is not circumscribed, as in the ordinary form, but shows a remarkable tendency to spread to the face and scalp, or downwards towards the clavicle. Vesicles form over the brawny swelling, especially on its mucous surface; they rapidly become purulent, and burst, disclosing the sloughy tissue bathed in pus. Infective phlebitis, embolism, and general infection are very common. If the jugular veins are involved, secondary centres may form in the lungs; or if the facial vein inflames, retrograde phlebitis may give rise to meningitis. Chills, or a decided rigor, with high fever and marked typhoid symptoms, are present in all cases, and death usually results from exhaustion or general infection in four or five days or less. The disease must be distinguished from malignant pustule, in which the swelling is localised, is covered with a brown *dry* slough, and is *surrounded*—not covered—by a ring of vesicles (see p. 114).

Treatment.—As for carbuncle.

MALIGNANT PUSTULE—LOCAL OR EXTERNAL ANTHRAX

Etiology.—Anthrax may occur as a local or general disease, the latter originating by inoculation through the pulmonary or intestinal mucous membrane, or following the local affection of the skin.

Malignant pustule is dependent upon local infection, with the bacillus anthracis at the seat of a wound, often merely an insect bite. It may probably also occur through the sebaceous or sweat-glands, or the hair-follicles. For obvious reasons, it is most usually met with on exposed parts—the face, hands, and forearms. Anthrax is common in all herbivorous animals, especially horned cattle, and in this country inoculation occurs in the case of those engaged in the preparation of hides or fleeces imported from infected

districts. Nearly all animals can be inoculated, but many are refractory.

The *bacillus anthracis* is a non-motile, rod-shaped, aërobic organism, with cup-like ends. In the living body it multiplies by fission only, but externally by spores also.

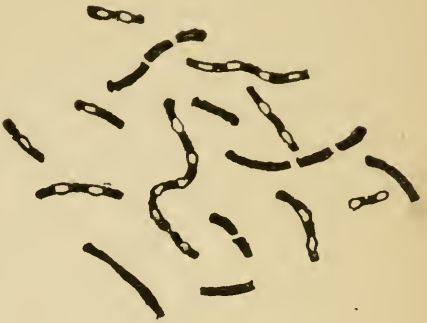

FIG. 25.—Anthrax bacilli and spores.

In cultures the bacilli grow into long filaments, and form interlacing bundles, but this does not occur in the body.

The spores are extremely resistant to injurious influences, but may be killed by boiling.

The bacilli spread by the lymphatics and blood-stream, and are found in great numbers in the capillaries.

The toxines are an albumose, a peptone, and an alkaloidal base. Leucin and tyrosin are also found. The organism can be attenuated by repeated inoculation from animal to animal, and a protective serum is thus obtained. Protection lasts, however, only a short time.

Signs and symptoms.—After inoculation there is an incubative stage of from some hours to three to four days, the duration depending on the dose and virulence of the organism and the predisposition or otherwise of the host. At the point of inoculation a small red pimple forms, accompanied by itching and heat, but pain is absent throughout the disease. In a few hours the pimple spreads and develops into a red, indurated, raised carbuncular patch, surrounded for some distance by inflammatory œdema and infiltration of the tissues, which may be shot with petechial hæmorrhages.

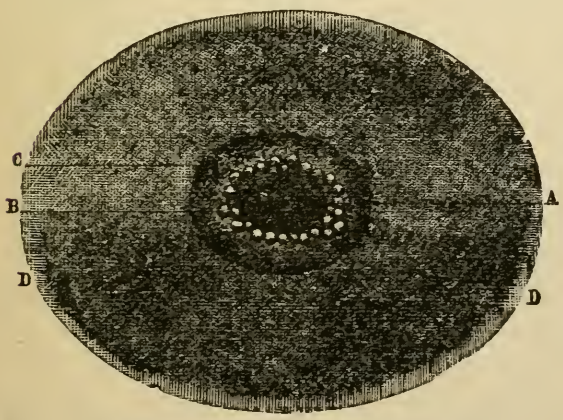

FIG. 26.—Malignant pustule. *A*, central slough; *B*, ring of vesicles; *C*, area of induration; *D*, area of surrounding congestion. (Follin.)

Lymphangitis and lymphadenitis are excited, the former

indicated by red and tender lines running from the pustule towards the nearest lymphatic glands, which are enlarged and tender.

Soon after the appearance of the pimple a small vesicle containing sanious fluid forms on it, and others make their appearance in the centre of the increasing patch. These vesicles may rupture or dry up. The skin beneath is at first a purplish colour, but soon dries, and turns brown or black. This gangrenous patch, with its adjacent inflammatory zone, increases in area, and is surrounded by a circle of vesicles similar to those which form over the sloughing skin (Fig. 26, p. 113). The slough extends to the connective tissue, but no suppuration occurs until it begins to separate. This absence of suppuration is very characteristic, affording marked contrast to what is seen in carbuncle. The sloughy tissues are blood-stained, as extravasation is a common feature of the lesion.

The inflammatory reaction round the pustule is much more marked in a few hours than at first, and serves to limit the process. Bacilli are found in the contents of the vesicles and in the superficial lymphatics, but not in the blood unless general infection ensues; nor are they found in the slough, having probably been killed and disintegrated so as to become indistinguishable.

Constitutional symptoms may be entirely absent, and the patient, unmindful of the local condition, continues his employment; more usually there is some rise of temperature with general malaise. If general infection ensues, severe symptoms of general anthrax, referable to the lungs or intestinal tract, make their appearance; marked dyspnœa, vomiting, bloody diarrhœa, and cardiac failure being the most prominent. For a full account of general anthrax the reader is referred to a work on Medicine.

Diagnosis.—Malignant pustule must be distinguished from carbuncle and facial carbuncle. The chief points indicative of malignant pustule are the patient's exposure to infection from the nature of his employment, the painless, non-suppurative course, the presence of the vesicles in the centre and round the dry brown slough, and the mildness of the general symptoms. The discovery of the specific micro-organism in the discharge by microscopic examination, or by inoculation of a predisposed animal is conclusive proof, but is rarely necessary.

Prognosis.—The prognosis in malignant pustule is good, provided it be detected early before signs of general anthrax are present and is energetically treated. If general infection has occurred, the patient's chances are small.

Spontaneous cure has been known, but must by no means be relied on to the exclusion of treatment.

The surrounding inflammatory œdema is on account of its situation, *e.g.* the neck, sometimes a danger by its possible involvement of specially important parts, such as the glottis.

Treatment must be energetic and early adopted. The pustule must be completely excised and the wound cauterised either with nitric acid, chloride of zinc paste, or the actual cautery. During excision free irrigation with 1 : 2000 mercury solution is advisable in order to wash away the blood and prevent the bacilli gaining entrance to the vessels, as general infection may be thereby occasioned. The veins leading from the part may be compressed with a like object, but no compression should be exercised on the pustule itself; the wound must be dressed with double cyanide gauze and mercuric cotton. The surrounding cellulitis will usually subside when the pustule has been removed ; incisions may be necessary for threatened suppuration, which, however, is rare.

The general treatment consists in the use of stimulants, quinine, and good diet, as in the infective processes generally.

Ipecacuanha has been shown to possess germicidal properties as regards the B. anthracis, but not as regards the spores. This, however, does not matter from a clinical point of view, since the latter are not present in the body. The powdered drug should be given in five or ten grain doses with a little milk ; the dose is to be repeated every six hours.

CANCRUM ORIS—NOMA VULVÆ

Seat.—Cancrum oris or noma is an acute, spreading, gangrenous inflammation met with in young children. It usually occurs in the groove between the lower jaw and the cheek, and may originate in ulceration of the gums. Occasionally it affects the mucous surface of the vulva.

Causes.—Noma usually attacks cachectic children between the ages of two and five years. The poor of large cities are specially liable to it. The subjects of noma are always in a state of marked debility either from starvation, ill-feeding, exposure, or faulty hygienic surroundings, or from a recent attack of one of the acute specifics, especially measles or scarlet fever.

The exciting cause is the presence of a slender bacillus which is found in the sloughy mass and in the margins of the inflammatory area.

Signs and symptoms.—The connective tissue beneath the mucous membrane is the seat of copious exudation which occasions much swelling. The cheek is swollen, red, hot, and brawny; the skin is stretched, shiny, and smooth, and in a few hours the most tense portion becomes black and gangrenous, and perforation of the cheek rapidly follows.

Movement of the jaw is limited, or it may be rigidly closed by the swelling. Salivation is profuse, and the saliva, mixed with discharge from the gangrenous mass, is horribly offensive. If the mouth be forced open, a foul, deep, and ragged ulcer covered with a dark slough is seen; the jaw may be denuded and the loosened teeth drop out. Pain is absent. Venous thrombosis with general infection frequently ensues, or the fatal termination may be due to septic bronchitis or pneumonia. The constitutional symptoms are severe; there is high fever with rapid pulse and signs of failing heart. In a few hours marked typhoid symptoms make their appearance, the child becomes apathetic and drowsy, and coma ushers in death, which usually occurs in from three to six days. Very few cases recover.

Noma of the vulva runs the same rapid course; it usually begins on one labium and extends to the other by contact. Death occurs from exhaustion or general infection.

Treatment is the same whether the disease attacks the mouth or vulva; to be effectual, it must be prompt and thorough.

The child must be anæsthetised, and the seat of the disease (as limited by the induration) must be thoroughly cut away by the knife, scissors, and sharp-spoon so that the whole area of infection is removed. As soon as this has been done the tissues should be thoroughly cauterised with strong nitric acid and the wound packed with gauze saturated with iodoform emulsion.

The mouth should be irrigated with Condy's fluid or chlorate of potash solution (gr. 20 ad ℥i) every hour. If the disease reappears the operation must be repeated. Nutritious fluid food and stimulants, with the internal administration of chlorate of potash, are the general measures to be adopted.

HOSPITAL GANGRENE

Hospital gangrene, regarded by many as identical with phagedæna, is, owing to improved sanitation and antiseptics, practically a thing of the past in civil practice. Formerly it was one of the most formidable dangers of military campaigns; in the Crimea this

disease proved most disastrous, and is said to have killed more men than did the Russian bullets. In modern campaigns it is very rare.

Hospital gangrene is distinguishable from phagedæna by its greater rapidity of spread, its more highly contagious nature, and its higher rate of mortality; phagedæna is not so virulently contagious, and occasionally ceases spontaneously or under mild treatment. No doubt all grades of severity are met with, so that the two diseases merge one into the other.

Causation.—Hospital gangrene is associated with the presence of staphylococci and streptococci, and Koch has, by inoculation of mice with the latter, induced a gangrenous affection identical in its general features with hospital gangrene. It is highly contagious. Bad hygienic surroundings, especially the overcrowding of surgical patients, are most potent predisposing causes. The poison may be conveyed to the patient by instruments, sponges, flies, etc.

Symptoms.—Hospital gangrene usually attacks recent wounds. It is ushered in by a feeling of weight and severe burning pain in the part. The surface of the wound inflames, and is covered by a thick, pulpy, loosely adherent, dirty black slough, which is usually separated from the inflamed margin. The surrounding tissues are swollen, livid, œdematous, and inflamed, and speedily die by extension of the process. The edges of the ulcer are steep, everted, ragged, and often undermined; when cut into, the infiltrated tissues are said to look like pork. The gangrene spreads superficially and deeply, gradually destroying muscles, vessels, etc., laying bare bones and opening joints. The vessels usually escape so long as the sloughs are adherent, but when separation begins, profuse and fatal hæmorrhage may occur. The discharge from the gangrenous area is horribly fœtid, of a dark colour, and often bloody.

In less formidable cases the gangrene does not extend so deeply, but covers a larger area; it is then not so fatal.

Restlessness, sleeplessness, general asthenia, and nervous prostration with high fever, are the leading constitutional features. Typhoid symptoms soon make their appearance; delirium sets in and deepens into coma as the end approaches.

Prognosis.—Nearly all cases of genuine hospital gangrene succumb. It is stated that at the siege of Sevastopol no person who was attacked by hospital gangrene was known to recover.

If the process be temporarily arrested, a relapse may occur in a few days and carry off the patient.

Treatment.—The patient and those nursing him must be rigidly isolated. All dressings should be burnt in the room, and

the instruments used must be thoroughly sterilised. Free stimulation and large doses of opium and quinine must be prescribed. Locally, the most drastic measures are the only ones of any avail. The patient must be anæsthetised and the gangrenous tissues freely removed by the sharp-spoon, care being taken that no large vessel is opened. The surface must then be rubbed over with the actual cautery, or treated with pure nitric acid or chloride of zinc paste, or it may be covered with Ricord's paste (sulphuric acid and willow charcoal in equal parts). This paste dries on the surface, and in from twelve to thirty-six hours can be easily removed, leaving a healthy surface beneath; it must be reapplied if necessary. If recovery ensues, the wound will heal by granulation. If there is a relapse, the treatment must be repeated.

Amputation may be required in case of relapse, or if a large vessel gives way and the bleeding cannot be controlled. It must, however, be remembered that amputation is a very serious measure, and that the patient is usually in too feeble a condition to withstand the shock; moreover, the gangrene may attack the stump. It is a desperate remedy, but under the circumstances mentioned will afford the patient a last chance.

SLOUGHING PHAGEDÆNA

Sloughing phagedæna closely resembles, if it is not identical with, the milder cases of hospital gangrene, but is far less contagious.

Causes.—In the main the causes are those of hospital gangrene. Sloughing phagedæna is particularly liable to occur in syphilitic and soft sores, especially if they are concealed under a tight prepuce.

Signs.—Assuming the disease to begin in a venereal sore, accompanied by phimosis, so that cleanliness cannot be properly secured, there will be considerable inflammatory swelling with acute pain. The prepuce is bulged by foul discharge, which drips away from the orifice, it becomes shiny, tense, and of a livid hue, and a small black area marks the onset of gangrene. The prepuce sloughs, and the glans penis escapes through the opening. Gangrenous ulceration rapidly extends in superficial area, but does not usually eat deeply; the skin of the penis, scrotum, perineum, groin, and abdomen may be completely destroyed. In the case of a woman recently under my care in the Westminster Hospital, a large gangrenous area resulted, involving the left side of the vulva, ischio-rectal fossa, perineum, nates, thigh, and abdominal wall.

The tissues are brawny and œdematous; the surface of the wound is covered by a dark or greenish slough, and the discharge is horribly offensive. This gangrenous process necessarily masks the original venereal sore.

Very often there are practically no constitutional symptoms beyond a slight degree of fever; sometimes they are similar to those of hospital gangrene.

Prognosis.—The rapidity and virulence of the process varies considerably; usually it spreads rapidly, at other times more slowly, and may even be spontaneously arrested. Recovery is the rule, but death may occur from exhaustion or septic poisoning.

Treatment.—The general treatment consists in the administration of opium and quinine, with stimulants if the state of the patient is low. Locally, the application of Ricord's paste is most beneficial; the sore should be dried, and the paste freely applied and allowed to dry on; it may be reapplied in twenty-four hours if there is any sign of a continuance of the disease. The prepuce, if tight, should be slit up to expose any sore beneath it.

In milder and less rapidly spreading cases, excellent results follow immersion in a boracic bath at a temperature of about 100° F. The patient may sit in this for seven or eight hours; the sitting should be repeated after an interval for rest, during which the parts should be freely dusted with iodoform and hot fomentations applied. If the patient can stand it, the sitting may be continuous. The baths should be continued until all sloughing ceases and the sore begins to heal.

Should phagedæna occur in a syphilitic sore mercury will be necessary, but if the patient is feeble it will be wiser to defer its use until the active stage of the sloughing has passed.

WOUND-DIPHTHERIA

Etiology.—Wound-diphtheria is probably closely allied to sloughing phagedæna, but has much less destructive tendencies, and does not show the same predilection for venereal sores. It usually occurs in dirty wounds and ulcers, and in cachectic patients, living under faulty hygienic surroundings. Micrococci are present in the exudate.

The condition sometimes assumes an acute form very like phagedæna.

Signs.—Lymph is effused on the surface of the wound, and the granulations show a retrograde tendency and may be the seat of

hæmorrhage. The exudate forms a tough, grayish-white membrane adherent over the surface of the wound, the edges of which may be slightly swollen and inflamed; as these break down, the ulcer increases in size. The process is generally very chronic and unaccompanied by constitutional symptoms, but should it assume a phagedænic type, the local condition is much more serious and constitutional symptoms may supervene. The contagiousness of this affection is slight.

Treatment.—The false membrane should be scraped away, the wound sharp-spooned, and cauterised with chloride of zinc paste or nitric acid. The general health must be improved by good food. Quinine and opium are very useful.

Healing is usually readily induced and the fear of recurrence under antiseptic treatment practically, *nil.*

SPREADING TRAUMATIC, OR EMPHYSEMATOUS GANGRENE

Emphysematous gangrene is a local infective process primarily attacking the subcutaneous connective tissue and causing acute inflammation and death of the affected parts. It spreads with great rapidity, subsequently affects the muscles and deeper structures, and usually proves fatal.

Causes.—The streptococcus pyogenes and a bacillus closely allied to, if not identical with that of malignant œdema, are the micro-organisms exciting this disease. The latter organism is abundant in surface soil and town mud. Chicken are specially susceptible to the organism which is often found in the earth of fowl-houses. It develops by spores outside, but not inside the body.

FIG. 27.
Bacilli of malignant œdema.

Emphysematous gangrene always occurs in connection with a wound, especially in drunkards, or in cachectic states such as are occasioned by diabetes or renal disease. Contused and lacerated wounds and those implicating joints or the medullary cavities of bones are specially liable to attack. It is very noticeable that wounds which have been contaminated with the mud of large cities, as is so often the case in compound fracture, are highly favourable seats of this disease.

Signs and symptoms.—Emphysematous gangrene attacks recent wounds, usually making its appearance about the second or

third day. Pain and heat in the part, with some constitutional disturbance, lead to an examination of the wound, which will be found swollen, inflamed, and discharging a brownish fœtid fluid suggestive of pent-up discharge. The swelling rapidly increases and extends up the limb, especially on the inner side where the cellular tissue is abundant and lax. The skin is of a livid purple hue, and the area of gangrene is marked by a livid blush. The infiltrated tissues are doughy and œdematous; they crepitate on pressure from the presence of gases evolved as the result of decomposition, and the action of the organisms; hydrogen and carburetted hydrogen are the most important. The skin is studded with blebs which contain fœtid, discoloured fluid, and the cuticle separates easily. The limit of the superficial blush does not quite correspond to the limit of the disease, as the connective tissue is primarily and the skin secondarily affected—a point to be remembered in amputating.

The gangrene extends with astonishing rapidity; commencing in the leg, it may reach the trunk within twelve hours. At the onset of the disease there may be shivering, or more rarely a distinct rigor, accompanied by a rise of temperature and marked constitutional disturbance due to the absorption of toxines from the decomposing and infected tissues. Later on the temperature may fall below the normal. The general symptoms are of the asthenic type; there is marked nervous prostration with delirium, deepening into coma and death within two or three days of the onset.

Prognosis.—The prognosis is extremely serious, especially if the disease approaches the trunk; the great majority of cases die.

Treatment.—Preventive.—All wounds which have been lacerated, contused, or contaminated with filth must be thoroughly cleaned with 1-20 carbolic acid. They must be enlarged if necessary, and the deeper parts reached by syringing, the syringe being armed with a piece of elastic tubing which can be insinuated among the torn tissues. If mud has been ground into the lacerated tissues the part should be cut away with scissors. Ample provision must be made for the escape of discharge, and rigid antisepsis observed.

Curative.—Early and high amputation is the only treatment offering a chance of success. The limb must be removed well above the blush, otherwise the incisions will pass through diseased connective tissue. After amputation the flaps should be examined and any œdematous and discoloured connective tissue must be removed with scissors; unless this be done the disease will continue

spreading. The stump should be thoroughly cleansed with 1-500 mercury solution.

Although amputation is the only remedy, it is a desperate resource, for the gangrene may and not infrequently does return in the flaps; even should this not occur, the shock of the operation may prove fatal to the patient in his critical condition. After the operation opium should be given and a stimulating plan of treatment adopted. Tubby has recorded a case in which amputation of the arm, followed by the injection of anti-streptococcus serum, was successful in spite of the fact that the disease had actually spread to the trunk before the amputation. In this case the bacillus of malignant œdema was not discovered, but the streptococcus longus was abundant.

CUTANEOUS ERYSIPELAS

Under the term erysipelas most surgical writers describe three conditions: (1) cutaneous erysipelas, (2) cellulo-cutaneous or phlegmonous erysipelas, and (3) cellulitis. If we are prepared to admit that these are all due to the same organism, the different clinical effects being dependent upon the conditions under which it acts, this classification is justifiable and right; but it would appear that the cutaneous form—erysipelas proper—is dependent upon a specific micro-organism, whereas the other two conditions are caused by ordinary pyogenic organisms or by mixed infection.

Fehleisen's streptococcus erysipelatis is identical in appearance and in its behaviour to stains and culture media with streptococcus pyogenes, and many assert that these organisms are one and the same. Similarity in these respects does not, however, necessarily imply identity of species, and Fehleisen claims that neither is the organism of cutaneous erysipelas capable of inducing cellulo-dermatitis or cellulitis, nor can cutaneous erysipelas be induced by the streptococcus pyogenes. These observations are, however, disputed by other authorities, who claim to have induced all three diseases by the same poison. Clinically, the occurrence of suppuration in cellulo-dermatitis and cellulitis and its absence in cutaneous erysipelas offers a striking contrast, but it must be remembered that the same organism may or may not induce suppuration according to circumstances. This clinical difference may depend on the greater virulence of the organism at any given time, or, what amounts to the same thing, on the lessened resistance of the tissues, or on the seat of inoculation. Meërovitch, who regards the streptococcus

erysipelatis as identical with streptococcus pyogenes, says that if the organism be injected into the muscular planes, it induces diffuse suppuration; if into joints, suppurative synovitis; and if into the peritoneal cavity, serous peritonitis which may become purulent. He, moreover, regards erysipelas as a general disease, and asserts that it only remains local so long as the organisms can be dealt with by the local phagocytes; when these prove inefficient, the organisms enter the blood-stream, and he claims to have proved that they may pass to the fœtus through the maternal blood. Cases of intra-uterine erysipelas have been recorded by others.

Meërovitch has succeeded in obtaining from cultures on raw meat a toxic alkaloid proving fatal to dogs.

In cutaneous erysipelas, cellulo-dermatitis, and cellulitis, the

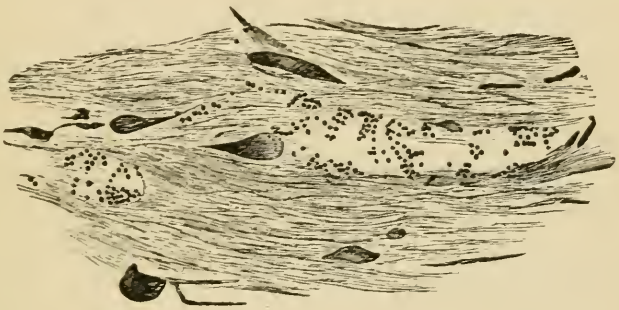

FIG. 28.—Section of skin at the spreading margin of the redness in erysipelas. A lymphatic vessel is seen containing micrococci, which are also spreading into the tissues around. (Watson Cheyne, from a photograph by Koch, ×700.)

disease spreads by the lymphatics, and excites inflammation of the vessel walls, coagulation of their contents, and lymphadenitis. In cutaneous erysipelas the organisms are found in the spreading margin of the blush and slightly in advance of it, but not in that part over which the margin has passed; in cellulitis and phlegmon they are present in all parts. Most authorities agree that the blood is not inoculable, and Fehleisen, having induced cutaneous erysipelas in a rabbit's ear by inoculation, arrested all constitutional symptoms by amputating the organ beyond the blush.

The local effects are due to the caustic action of the toxines, the constitutional symptoms to their absorption. At present it must remain uncertain whether Fehleisen's streptococcus erysipelatis is identical with the streptococcus pyogenes, and whether all three conditions usually described as erysipelas are identical as regards etiology; but it appears probable on the evidence that we should attribute cutaneous erysipelas to a distinct organism—S. erysipelatis;

cellulitis to S. pyogenes and other pyogenic organisms; and cellulo-dermatitis to mixed infection of S. erysipelatis and S. pyogenes.

Etiology.—In all cases the streptococcus erysipelatis gains entry through a wound, which may be so small as to be readily overlooked, and hence it has been asserted that erysipelas may arise idiopathically. Minute cracks, scratches, or eczematous patches are often the seats of infection about the face; these are frequently situated near the angle of the mouth, ala of the nose, inside the nostril or behind the ear. Any part of the body may be affected, but the head, neck, and especially the face are the favourite seats. New-born children may be infected through the umbilicus. Individual susceptibility is marked; and although one attack appears to confer immunity, this is of a very temporary nature, and repeated attacks are common.

Bad hygienic surroundings, chronic alcoholism, diabetes, kidney-disease, or impairment of the general health from any cause are important etiological factors. Erysipelas may assume a distinctly epidemic character, and is more common in spring and autumn and in damp and cold weather.

Constitutional symptoms are present a few hours before the appearance of the blush. The onset is sudden and marked by chills or a distinct rigor, the temperature rising to 103°-106° F. Frontal headache, nausea, vomiting, furred tongue, constipation, pains in the limbs, and general malaise are all present. The pulse is rapid, full and soft, and as nervous prostration sets in, its rapidity increases and it becomes feeble and small. Nervous prostration comes on rapidly and is often severe; delirium sets in at night, the tongue is dry and brown, the lips loaded with sordes, and constipation may be succeeded by offensive, sometimes bloody diarrhœa. Death may ensue from exhaustion or from some intercurrent mischief, especially pneumonia.

The local signs appear within a few hours of the constitutional disturbance. There is general superficial tenderness and tingling, with a feeling of stiffness of the skin, which pits slightly on pressure. The affected area is covered with a vivid red blush having a well-defined, slightly raised margin, in and a little beyond which the streptococci are abundantly found (Fig. 28, p. 123), but they are absent elsewhere.

The blush quickly disappears on pressure and as quickly returns; it disappears at death. Should recovery take place, the blush is replaced by yellowish discoloration; branny desquamation follows, accompanied by a falling of the hair, which, however, quickly grows again.

The lymphatics running from the parts are engorged and inflamed, and the neighbouring glands are swollen and tender. Suppuration does not occur, but loose bullæ containing clear or sanious serum often form on the surface. The bullæ may burst, or dry up and form dry scabs. There is considerable exudation into the subcutaneous connective tissue, which does not, however, participate in the inflammatory process. The degree of swelling thus induced depends on the laxity of the tissues; in the face it is very marked, closing the eyes and causing the most unsightly appearance. If there be a wound of any appreciable size, healing is arrested; the granulations are pale and flabby and undergo atrophy, and the surface, dry and glazed at first, becomes bathed in pus as inflammation sets in.

Diagnosis.—Simple erythema may readily be diagnosed from erysipelas by the absence of fever and general disturbance, and by the redness being diffuse and patchy, and unaccompanied by tenderness or pitting on pressure.

Prognosis.—Cutaneous erysipelas is not a dangerous affection in otherwise healthy patients, recovery taking place in from ten to twenty days. The aged, those addicted to alcohol, and the subjects of general disease—especially diabetes and renal mischief—are liable to suffer severely and may die. Cardiac failure, as evidenced by the rapidity and feebleness of the pulse, high fever, persistent delirium, and profuse diarrhœa are ominous signs.

Erysipelas about the orbit or in connection with scalp wounds may spread to the meninges, and œdema of the glottis is to be feared when the fauces are attacked. Hypostatic pulmonary congestion and pneumonia are serious and not uncommon complications, especially in old people. Recurrence of erysipelas is common; after repeated attacks (especially in the face) the skin becomes thickened, hard, and inelastic, and the change may give rise to marked and permanent alteration in the personal appearance.

Treatment.—The patient must be rigidly isolated. Constitutional treatment must be directed to preventing and combating the prostration induced by the disease. The strength must be maintained by good, nutritious diet, with stimulants: port wine, champagne, and brandy being the best. The action of the skin, kidneys, and bowels must be regulated. Ammonia, quinine, and iron are the most useful drugs. Iron is thought by some to have a specific effect and may be given in half-drachm doses of the tincture every four hours. Aconite, pilocarpine, and salicylate of soda have their advocates. Numerous local applications have been vaunted by some, condemned by others. In facial erysipelas the parts must be

freely dusted with starch-powder or oxide of zinc, and should be kept from the air by a thick mask of cotton wadding. Some surgeons speak highly of the application of a 30 per cent solution of tincture of iron, which should be freely rubbed into the skin; others recommend the use of the solid nitrate of silver to the margin of the blush; but it seems very doubtful if either of these methods has any beneficial influence, either in arresting the spread of the disease or of a curative nature. German surgeons extol the virtues of equal parts of ichthyol and vaseline rubbed into the skin twice daily, and claim that it cuts short the disease in three days. A 10 per cent solution of ichthyol in collodion, painted over the blush and some distance beyond it, is also recommended. Koch advises that the parts and the skin round should be anointed twice daily with creolin 1, iodoform 4, and lanolin 10 parts; while Rosenbach applies daily a 5 per cent solution of carbolic acid in absolute alcohol. Hallopean asserts that the disease may be cured in four days by applying cotton wadding soaked in 5 per cent solution of salicylate of soda. Before any local application is applied the parts should be thoroughly cleaned with 1:1000 mercury solution.

Kroell, Woelfler, and others adopt the so-called mechanical treatment with the view of preventing the organisms passing along the lymphatics. The part is encircled by a bandage or adhesive strapping about 2 cm. beyond the blush, which does not spread beyond it; the encircling band should be retained for three or four days after the temperature has fallen to the normal.

In view of the fact that the streptococci are confined to the margin of the blush and about 2 cm. beyond it, and that it is readily killed by 1-30 carbolic solution, Hueter advises subcutaneous injection of the latter in the area indicated; numerous small punctures must be made, and it must be borne in mind that poisonous effects may follow the injection of large quantities. As a modification of this plan, Kraske first scarifies the skin at the margin of the blush and then rubs in the carbolic solution.

If bullæ form the fluid may be evacuated, but the epithelium, which serves as a protection to the papillæ, should not be removed.

Any wound which may be present should be thoroughly cleaned and free drainage must be provided.

An antitoxin serum has been prepared by Marmorek, injection of which is said to bring about cure, even in severe cases; at present the cases reported are not sufficiently numerous to afford us grounds for speaking positively as to the results of this treatment. It is, however, worthy of trial (see p. 215).

During convalescence the diet should be generous and should include stout or port wine. Tonics, especially the mineral acids and vegetable bitters with iron and quinine, and plenty of fresh air, are the best means of combating the anæmia and weakness left by the disease.

CELLULO-DERMATITIS (CELLULO-CUTANEOUS OR PHLEGMONOUS ERYSIPELAS)

Etiology.—As stated on p. 122, there are grounds for believing that this disease is distinct from erysipelas, and is probably dependent on mixed infection with the streptococcus erysipelatis and streptococcus pyogenes; but at present opinions conflict and no positive proof on either side is forthcoming.

Cellulo-dermatitis attacks recent wounds, usually poisoned from the first, which implicate the subcutaneous tissue or penetrate the intermuscular planes. It is not highly contagious, and if due care be taken to prevent inoculation by means of sponges, instruments, the hands, etc., there is no danger of the disease spreading among surgical patients. It especially attacks drunkards and the cachectic.

Local signs.—The inflammation begins in the subcutaneous tissue and quickly implicates the skin. The connective tissue is infiltrated with exudation, which causes great œdema and swelling, and the parts pit readily on pressure. There is considerable burning, throbbing pain from pressure on the nerves. The skin is tense and covered by a deep red blush, wanting, however, the distinct margin seen in cutaneous erysipelas. The lymphatic vessels and glands may, but do not necessarily, participate in the process. With the increase of the exudation tension becomes greater; the natural folds and wrinkles of the skin are lost, it becomes brawny, smooth, shiny, and of a dusky, livid colour. The disease spreads most rapidly on the inner aspect of the limb, where the cellular tissue is most abundant and loose.

Diffuse suppuration with sloughing of the connective tissue and localised gangrene of the skin, preceded by the formation of blebs, occurs in from three to five days of the onset. The amount of destruction varies with the intensity of the process and the promptness of treatment. If incisions are made early tension is quickly relieved, the pain diminishes or disappears, and sloughing of the skin is averted or at least reduced to a minimum. Unless the original wound has opened the deep fascia, the intermuscular planes are not involved; but in such a case the destruction may be very

widespread, the muscles, nerves, and vessels being more or less completely dissected out. Even in the worst cases hæmorrhage is a rare event, since the large vessels escape destruction and the smaller ones are thrombosed as the result of inflammation of their walls.

If an incision be made into the brawny area it gapes widely, disclosing the connective tissue infiltrated with ashen-gray or purulent exudation, and large yellow sloughs bathed in pus.

Should the part recover, the sloughs slowly separate, and healing takes place by granulation. The new scar-tissue may cause considerable impairment of motion by matting together the muscles, should their cellular planes have been affected.

Constitutional symptoms.—High fever, with asthenic symptoms and prostration, is the rule. The fever may reach 105° F. and be ushered in by chills or a rigor. Headache, anorexia, thirst, vomiting, and delirium at night are present. Sometimes—especially in drunkards—delirium is noisy and constant, and delirium tremens may supervene. The tongue is furred, and later on dry, cracked, and loaded with sordes.

Constipation is usual, but diarrhœa sets in in bad cases. The pulse is at first rapid and full, but becomes small and feeble as nervous prostration deepens. In fatal cases typhoid symptoms make their appearance and coma precedes death.

Prognosis.—The prognosis of cellulo-dermatitis is always grave, since it occurs chiefly in drunkards and those broken down by disease. It is most serious when attacking the head and neck and other regions rich in cellular tissue, and when it spreads to deep parts, *e.g.* the mediastinum, where it is beyond the reach of surgical treatment. High fever, persistent delirium, diarrhœa, and cardiac failure are serious signs. Hypostatic congestion and pneumonia or general septic infection may prove fatal.

The prognosis as to the ultimate utility of the part depends upon the extent and depth of the process. Matting of the tendons and muscles may be more or less permanent, or the destruction may be so widespread that amputation is the only resource.

Treatment.—Although isolation of the patient is not necessary, every care must be taken that the contagion is not spread from patient to patient in a hospital ward.

The general treatment, with the exception of the administration of iron, is practically that given under cutaneous erysipelas (p. 125). The wound should be thoroughly cleaned, efficient drainage provided, and tension relieved by the removal of any sutures which

may have been used. Moist heat, applied by hot boracic fomentations, or by immersion in the hot boracic bath, may cut short the process and avert suppuration. The part should be elevated if possible.

As soon as pitting gives place to brawniness, tension must be relieved by free incisions. These should be numerous and of sufficient length, and made, if possible, in situations devoid of large veins, as the loss of blood may be a serious matter in a debilitated patient. Any divided vessels should be ligatured and profuse oozing arrested by temporary plugging with antiseptic gauze. If the wound and tissues exposed by the incisions be swabbed over with pure carbolic acid, the further spread of the mischief is arrested. The separation of the sloughs must be encouraged by the continued application of heat and moisture. When granulation is fully established the wounds should be dressed with some simple lotions, such as boracic acid or red wash. During the healing process, gentle passive motion and massage will do much to obviate matting of the tendons and muscles, and to restore the suppleness of the parts.

If the destruction of the limb is so great as to make amputation advisable, this should not be performed until the disease has been arrested and the patient has so far recovered from its effects that he can stand the shock of the operation.

Treatment with anti-streptococcus serum has apparently been beneficial (see p. 215).

CELLULITIS (CELLULAR ERYSIPELAS)

Etiology.—The organism usually present is the streptococcus pyogenes, but it is practically certain that this is by no means the only one capable of inducing cellulitis. The disease is especially likely to occur in poisoned wounds, and may follow the sting or bite of a poisonous insect. Drunkards and cachectic patients are especially liable to attack.

Local signs.—The pathological and clinical features of cellulitis are similar to those met with in cellulo-dermatitis, the only difference being that in the latter condition the skin is equally affected with the subcutaneous tissue, whereas in cellulitis it is secondarily involved from interference with its blood supply by means of the pressure exercised on the vessels by the inflammatory exudate.

Suppuration is less common in cellulitis than in cellulo-dermatitis, and the former may occur at a distance from the seat of

inoculation. In some cases cellulitis spreads rapidly, in others it remains more limited and is quickly subdued by treatment, or should suppuration occur, the pus remains localised.

The constitutional symptoms are similar to those of cellulo-dermatitis, but do not usually run so severe a course, especially if suppuration is averted.

Prognosis.—Cellulitis is not usually dangerous to life unless it occurs in a dangerous situation. The prognosis depends chiefly on the intensity of the process and the state of health of the patient.

Treatment.—That of cellulo-dermatitis.

CELLULITIS IN SPECIAL REGIONS

Cellulitis of the scalp may complicate dirty and ill-drained wounds. The inflammation occurs beneath and is limited by the cranial aponeurosis. There is considerable swelling and great pain, with puffiness and bagginess of the tissues. Extension to the meninges may occur through the emissary veins or through a fracture, and hence the prognosis is grave. The treatment consists in removal of the hair, the employment of antiseptics, and incisions made parallel to and between the main vessels and nerves, coupled with the application of hot boracic fomentations.

Cellulitis of the orbit may result from a wound or from suppurative panophthalmitis. There is considerable pain, with swelling and closure of the lids, chemosis of the conjunctiva, and proptosis. Unless tension be relieved by timely incisions, the inflammation may extend backwards through the sphenoidal fissure, or by the ophthalmic veins to the cavernous sinus. The lid should be raised, a free incision made between it and the globe into the cellular tissue, and hot boracic fomentations applied.

Cellulitis of the neck—Angina Ludovici.—Cellulitis of the deep planes of the neck may result from ulceration or sloughing about the mouth or throat, or from extension of a suppurative process in connection with middle ear disease or dental caries. It may also occur in connection with strumous glands. The inflammation usually begins at the upper part of the neck and extends downwards. In the worst cases it may spread to the superior mediastinum. Owing to the density of the deep cervical fascia the tension and pain are severe, and the former may cause difficulty in swallowing and respiration. Extension to the larynx and œdema of the glottis constitutes a formidable danger. Prompt incisions must be made and so placed that no important structure is

damaged. Laryngotomy may be required if the disease spreads to the larynx.

Cellulitis sometimes begins in the cellular tissue round the base of the tongue, and in such cases the swelling is centrally placed and symmetrical beneath the jaw. There is considerable danger of extension to the larynx, causing sudden and fatal œdema; this may, however, usually be averted by an early and deep median incision.

Pelvic cellulitis most usually occurs in connection with abortion or parturition. It may complicate gonorrhœa or follow instrumentation of the uterus, and may also occur after operations about the perineum. In women the poison usually gains access through abrasions or wounds of the cervix uteri; the process begins in the peri-uterine connective tissue, and spreads by means of the rich lymphatic network of the pelvis. It is often complicated by pelvic peritonitis. Pelvic cellulitis occasionally occurs in men, especially in connection with operations about the pelvic viscera.

There are the usual signs of fever. Pain above the pubes is complained of, often referred to one or other side, and on vaginal examination an indurated, acutely tender patch is felt, displacing and fixing the uterus. Suppuration does not usually occur; should it do so the patch softens, the general symptoms deepen, and the fever becomes remittent. The abscess may burst into the vagina or rectum, open on the groin, or escape by the sacro-sciatic notch. Very rarely the pelvic abscess bursts into the bladder or general peritoneal cavity. Unless great care be taken to prevent it, putrefaction of the contents of the abscess may occur and seriously add to the patient's danger. It sometimes happens that a dense mass of cicatricial tissue is left, which by involving the ureter may lead to secondary hydronephrosis.

Treatment.—To promote absorption and prevent suppuration the patient must be kept at rest, with hot fomentations applied to the hypogastrium. The bowels should be kept acting, and warm, antiseptic vaginal douches used frequently. If suppuration occurs the abscess must be freely opened and drained.

RABIES (HYDROPHOBIA)

Etiology.—Rabies is always communicated from a rabid animal by inoculation, and never arises spontaneously. It is, doubtless, dependent on a micro-organism; but none has so far been discovered. The poison is chiefly present in the saliva and

central nervous system, but exists in all tissues and secretions. It is attenuated by moderate, and destroyed by prolonged desiccation. It by no means follows that a bite from a rabid animal is followed by rabies; indeed, about 50 per cent escape. This immunity depends chiefly upon the situation and extent of the wound. Bites on the face, hands, or exposed parts, especially if multiple, are more dangerous than those on protected parts, where the injuries are not so extensive, and where entrance of the saliva is in some measure prevented by the clothes. Individual immunity may also have some bearing on the point. Dogs, wolves, jackals, foxes, and cats are the animals chiefly subject to rabies; but it may be communicated to all—even to birds. In this country dogs are the usual source of infection.

Symptoms.—The **incubative stage** varies within wide limits. The symptoms generally make their appearance during the second month after inoculation, but may be postponed for as long as two years. The wound, which has usually soundly healed, and has given no trouble, often becomes the seat of prodromal pains and tinglings which sometimes radiate along the nerves. The scar may reopen, or be studded with vesicles. The patient is restless, depressed, and apprehensive of coming evil. He is often reserved about the fact of inoculation, but shows a repugnance to fluid, with perhaps difficulty in swallowing it, which he tries to overcome. Hydrophobia in a marked form is not yet present. There is anorexia with vomiting. This prodromal stage may be absent or may last for from one to seven days; it suddenly passes into the fully developed disease.

The **acute symptoms** begin by violent convulsions of the deglutition and respiratory muscles. The convulsions are spasmodic, not tetanic; they may be momentary, or may last for some minutes, and are repeated at longer or shorter intervals, the spasm being frequently determined by some trivial cause, such as a noise (especially the sound of running water), the sight of water, a slight draught, or a mere touch of the skin. At the onset of an attack, the sudden contraction of the diaphragm and muscles of respiration may occasion a cry or a series of short, quick, sobbing sounds.

During the paroxysm the patient's appearance is most distressing and terrifying to the bystanders. He suddenly springs up in bed, clutches at his throat, and tosses his arms and head about; the face is pale and terror-stricken, the pupils dilated, and the skin often bathed in sweat. Viscid saliva accumulates in and dribbles from the mouth. This causes the patient much annoyance, and in his

efforts to get rid of it he may spit it in all directions, and on his attendants. This saliva is contagious.

Painful erections, satyriasis, and emissions are common. The most marked and predominant symptom is hydrophobia, and from this fact the disease takes its name. In the early stages the patient may make heroic efforts to overcome his dread of water; but as soon as the fluid touches his lips, in spite of his thirst he flings the glass from him and a paroxysm comes on. The sound of running water, or the mere sight of fluid, is equally disturbing. In rare cases hydrophobia is absent, so that the patient will make every endeavour to assuage his thirst, but this quite ineffectually, owing to the supervention of spasm.

The mental balance is much disturbed. In all cases there is evident terror, extreme restlessness, and mental agitation. The patient talks volubly, quickly, and with marked agitation; he frequently shouts and cries out, and is suspicious of all who come near him. Sometimes he constantly refers to the animal that bit him, and may himself try to bite those around him. Acute mania of a violent form is sometimes present. The mental excitement may temporarily subside and give place to quietude and rationalism, only to recur with greater violence.

The temperature may be normal throughout, or rise one or two degrees; the pulse, after a short time, is feeble and rapid. Towards the end general muscular exhaustion becomes marked, and the patient may develop the paralytic signs met with in the "dumb" rabies of animals.

Sudden death may ensue from cardiac failure, or spasm of the glottis and suffocation.

Diagnosis.—The diagnosis from tetanus is easy. In that disease the incubative period is short, the muscles are tetanised, the intellect remains clear, there is no viscid saliva from the mouth, and hydrophobia is absent.

When any one has been bitten by a dog supposed to be rabid, the animal should by no means be killed, but safely secured, and watched for weeks if necessary. If it has been killed, it should, if possible, be sent to an institution where inoculation may be employed to confirm the diagnosis of rabies.

Rabies in the dog is characterised by sudden alteration in his temper and habits. He is subject to paroxysms of fury, snaps at his fellows or at imaginary objects, and bites his kennel, straw, or chain; he is very restless, and moves about in an aimless way regardless of things which would attract his attention if in a state of health; he

is languid in spite of his restlessness, and hides himself away in darkened corners. There is no hydrophobia, and the animal will greedily lave his muzzle (dripping tenacious, and highly infective saliva) in water, but is unable to drink. He rapidly emaciates, his appetite is perverted, and he will swallow all sorts of rubbish. He is generally "humped up," with his back curved and his tail drooped. His cry is characteristically ringing, high-pitched, and croupy. Towards the end he passes into the paralytic stage.

Paralytic or dumb rabies may be present from the first, and is characterised by paralysis of the hind-quarters and of the jaw, so that the dog cannot bite or bark; the tongue protrudes, and saliva dribbles from the mouth. The animal is lethargic and quiet. Death ensues within a week.

Prognosis.—Rabies is fatal to man in from two to seven days. Death may be due to exhaustion or suffocation.

Post-mortem appearances.—The chief signs met with in rabies are congestion and incipient inflammation of the pharynx, salivary glands, and certain parts of the nervous system. In the spinal cord and medulla, especially about the deglutition and respiratory centres, viz. the roots of the glossopharyngeal, vagus, and hypoglossal nerves, there is congestion of the vessels, with exudation of leucocytes round their sheaths accompanied by minute hæmorrhages. The nerve-ganglion cells are degenerated.

Treatment.—**Preventive.**—When a person has been bitten by an animal supposed to be rabid, he should without delay be treated by Pasteur's inoculation method. Pending this the wound, which may be freely sucked by the patient, should be thoroughly cauterised, or completely excised. The "simple method" of Pasteur is applicable to all cases where the bite has been through the clothes, and to slight wounds on the soft parts. The "intensive" method is advisable in more severe cases. Two inoculations of varying strength are made daily for a fortnight or more. The injections are made into the subcutaneous tissue of the loins or abdomen, and do not give rise to local or general disturbance. The vaccine is prepared from dried pieces of the spinal cord of inoculated rabbits. The longer these have been desiccated the less virulent they are, and hence the dose may be increased by using cords which have been dried for shorter periods. The following table will show the method of treatment on each successive day of inoculation:—

Simple Method. Days of Drying the Cord.		Day of Treatment.	Intensive Method. Days of Drying the Cord.			
14	13	1	14	13	12	11
12	11	2	10	9	8	7
10	9	3		6	6	
8	7	4		5		
6	6	5		5		
5		6		4		
5		7		3		
4		8		4		
3		9		3		
5		10		5		
5		11		5		
4		12		4		
4		13		4		
3		14		3		
3		15		3		
		16		Rest		
		17		Rest		
		18		5		
		19		4		
		20		3		

Each injection consists of 5 c.cm. of spinal cord crushed in 2 c.cm. of sterile beef-tea. On the days when two strengths are given, one is injected in the morning, the other and stronger in the evening.

Curative.—When rabies has actually come on, treatment is of little avail, but the intensive method should if possible be adopted. Mercurial inunction is said to have been successful; but more reliance is placed on curari, physostigmine, chloral, morphia, and chloroform. The two last may be used to quiet the patient if there is much mental excitement, but chloral is most efficacious in lessening the spasm. Curari is well tolerated by rabid patients, and may be given in doses of one-eighth of a grain every fifteen or twenty minutes until benefit is derived; but care must be taken that muscular paralysis is not induced, or the breathing will stop. The patient should be kept quiet in a darkened room, and all causes likely to induce a paroxysm must be carefully avoided. Restraint by the strait waistcoat may be necessary. Plenty of food must be given by the rectum —under chloroform, if necessary. The attendants should be very careful that they do not inoculate themselves with the saliva or other secretions.

TETANUS

Etiology.—Tetanus is much more common in the Tropics than in temperate climates. It may occur at any age, and is more frequent in men than in women (4 : 1), and especially in the debilitated. Inoculation occurs through a wound ; those which have been fouled with soil containing the bacillus or into which splinters of wood, etc., harbouring it have entered, are specially liable to infection.

The *bacillus tetani* is present in surface soil, especially in stables. It is an obligate anaërobe (see p. 91), and this is probably the reason why it does not enter the blood but remains strictly localised to the seat of inoculation, where it develops and pours its toxines into the circulation. It is never found in the blood or lymph or at a distance from the wound. The organism is small and develops a spore at one end, so that it resembles a round-headed nail or small drum-stick. These spores are very resistant, but boiling kills them in a few minutes. Kitasato has isolated tetanine, tetano-toxine, and tetanus tox-albumose from pure cultures ; they are all toxic, but the last is the most virulent. The antitoxin is referred to under Treatment.

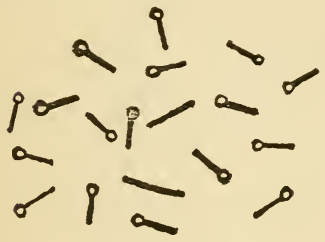

FIG. 29.—Tetanus bacilli and spores.

Symptoms.—Tetanus usually supervenes in from three to five days after inoculation, but may occur earlier or be postponed for a month or more. The onset is usually sudden, but sometimes more gradual, the patient complaining of general malaise and showing mental uneasiness. Neuralgic pain in the wound may, though rarely does, occur. With the onset of the disease the patient experiences stiffness and cramp about the muscles of the neck, face, and jaws, with difficulty in opening the mouth. This is quickly followed by tetanic spasm of the muscles, especially those supplied by the fifth, seventh, and eleventh cranial nerves. The jaw is tightly clenched (lock-jaw or trismus) ; the natural lines about the face and forehead are deepened, and the patient has a prematurely aged look ; the angles of the mouth are retracted into a fixed mirthless smile (risus sardonicus). The sterno-mastoids and muscles of respiration are strongly contracted, and thus produce difficulty in breathing, which is short and catchy. Occasionally the spasm is confined to the muscles mentioned, but

more usually the spinal nerves become affected and tetanic spasms are almost universal; the legs and forearms generally escape. The respiratory difficulty may be marked; the voice is feeble and more or less cyanosis is induced. Epigastric pain is usually present, and is probably due to spasm of the diaphragm. The tetanus is constant, but is temporarily increased by violent and extremely painful paroxysms of a cramp-like nature. These may be induced by the slightest cause, such as a draught of cold air, a noise, shaking the bed or merely touching the skin; such paroxysms are very painful, may last a few seconds or many minutes, and are repeated at long or short intervals.

During a paroxysm the whole body is usually arched backwards through violent contraction of the dorsal muscles (opisthotonos); the neck is hyper-extended, the occiput being approximated to the interscapular region, and the face looking vertically upwards; the chest is pushed forwards, and is in the position of expiration; the abdomen is flat and hard as a board, and the sections of the recti stand prominently out. The muscular contraction may be so violent that the muscles are ruptured, and considerable hæmorrhage occurs into their substance; occasionally bones (especially the ribs) are broken. The contracted muscles are dense and hard to the touch.

Sleeplessness is a marked feature of the disease, but the intellectual faculties remain clear and unclouded to the last. If sleep is induced by treatment the muscles partially relax, but contract again with waking.

Fever may be absent throughout the disease, but it usually appears in a few hours and is often high; hyperpyrexia is not uncommon. The fever is probably dependent upon irritation of the heat centres by the toxines.

Sweating is often profuse, but may be absent. Towards the end of the disease the pulse becomes irregular, very rapid, and is counted with difficulty.

Diagnosis.—Lock-jaw is the earliest and one of the most prominent signs of tetanus, and may, before other symptoms have developed, be confounded with the temporary closure of the jaws produced by difficulty in cutting a misplaced wisdom tooth. The presence of a recent wound, coupled with the evident affection of the facial and neck muscles, and in some cases the age of the patient, are sufficiently diagnostic. Hysteria occasionally simulates tetanus, but the distinction is easy.

Rabies and tetanus can hardly be mistaken (p. 133). Poison-

ing by strychnia presents signs very like tetanus, but the absence of a wound, the suddenness of the onset, and the fact that the muscular contractions are clonic and not tonic, and that there are periods of complete muscular relaxation, will prevent any mistake.

Prognosis.—The shorter the incubative period the more violent is the tetanus, and the more quickly will a fatal result ensue. Death usually occurs in from three to seven days, and should the patient survive beyond this time he may eventually recover. The fatal termination may be due to exhaustion, spasm of the glottis or respiratory muscles, or to cardiac spasm or paralysis; the latter ensuing in consequence of the greatly increased peripheral resistance to the circulation induced by the powerful tetanic contractions.

Treatment.—The wound must be freely opened up if necessary and thoroughly cleaned with 1 : 1000 mercuric solution, free drainage being ensured. If a foreign body is suspected it should be sought for and removed.

Amputation is usually discredited by surgeons, but it may here be noted that as the bacillus is strictly confined to the wound, amputation must effectually remove it and thereby prevent any fresh absorption of its toxines. The bowels must be opened and kept acting, and the action of the skin and kidneys promoted by diaphoretics and diuretics. Every known anti-spasmodic has been used, but without any very marked effect, except in chronic cases. Chloral, physostigmine, morphia, and chloroform are those on which some reliance can be placed. The patient is very tolerant of these drugs, and they must be given in large doses and pushed until some distinct physiological effect is produced. Violent and agonising paroxysms are best controlled by the inhalation of chloroform, and with the help of this drug the patient may be fed and stimulants administered through a stomach tube or by enemata. Baccelli strongly advocates the hypodermic injection of a 3 per cent solution of carbolic acid, and states that it promptly relieves the symptoms, and is more successful than any other treatment, including that by antitoxin. The drug must be freely given in doses of 3 or 4 centigrammes of the solution repeated several times during the twenty-four hours; its free use is especially recommended. Out of thirty-four cases treated by this method only one died.

Antitoxin injections.—The tetanus antitoxin serum (introduced by Tizzoni) is obtained from horses which have been artificially immunised by injections of toxine solution of gradually

increasing strength. Three or four days after the last inoculation the animal is bled by means of a cannula in the jugular vein; the blood is allowed to coagulate for twenty-four hours, and the antitoxin serum is then drained off. This serum is evaporated to dryness *in vacuo* over sulphuric acid, since heat destroys its properties. The powder thus obtained may be kept like any other drug in sealed tubes, and when required for use is dissolved in sterilised water without heat; 1 gramme may be dissolved in 5 or 10 c.cm. of water.

A gramme of the dried serum is equivalent to 10 c.cm. of the wet. In the treatment of tetanus the injections are made into the abdominal wall or thighs, or both. It is advisable to start the treatment with 20-40 c.cm. of the fluid serum (= 2-4 grammes dried), to be followed up by a quarter of that dose every six hours. The benefit is sometimes immediate, but the real value of the drug is open to question; many cases are certainly not appreciably relieved.

There seems, from perusal of the published cases, no doubt that acute cases are not so amenable as are chronic, and it must not be forgotten that 50 per cent of the latter recover in any case.

Roux and Borrel have successfully treated tetanus by intracerebral injection of the antitoxin. A small incision is made through the scalp and a hole made in the skull with a small drill in the centre of a line drawn from the outer angle of the orbit to the middle of a line from one auditory meatus to the other. A fine blunt needle is inserted into the brain for about 1½ to 2 inches, and .5 grain of dry antitoxin in 5 cm. of water is very slowly injected so that it may be absorbed. The operation is repeated on the other side and antitoxin is also given hypodermically. The object of this method is to immunise the nerve centres.

The antitoxin treatment should be combined with the administration of chloral, physostigmine, etc.

ACTINOMYCOSIS

Etiology.—Actinomycosis is a very chronic disease, due to the **actinomyces or ray fungus**, one of the Cladothricaciæ. The fungus occurs in the form of small grayish or sulphur-yellow nodules about the size of a hemp seed, each of which is a cluster of yet smaller masses. The difference in colour is a question of age, the yellow masses having undergone fatty degeneration.

Microscopically the fungus is seen to consist of a central felt-

work of branching, wavy filaments, and coccus-like spores; radiating from the central mass are filaments, which are often branched and clubbed at their extremities; these clubbed filaments are probably the degenerated ends of those forming the central network.

Fig. 30.—Actinomycosis hominis. (Ziegler.)

Boström has shown, by cultivation and inoculation, that the central filaments, and not the club-shaped radiating processes, are the true infective agents.

Actinomycosis is chiefly met with attacking the jaws and tongue of horned cattle, but is not directly communicated by them to man. Inoculation may occur through the respiratory or alimentary tracts, especially by the mouth and pharynx. Abrasions, ulcers, and carious teeth probably open up paths for infection.

The fungus is introduced by barley grains or straw. In man the liver is a common seat of the affection, inoculation being effected through the intestinal tract, especially the colon. The disease spreads by continuity of tissue and by the lymphatic paths.

Morbid anatomy and signs.—The disease is usually very chronic, and is characterised by the formation of a dense, slowly growing mass composed of round-celled infiltration, which subsequently undergoes fatty degeneration and necrosis. The granuloma is surrounded by more or less dense scar tissue, which may be calcified; the slowness of the growth, and the tendency to spontaneous arrest is directly proportional to the amount and density of the fibrous tissue. By gradual invasion of the tissues the granuloma increases in size and affects bones, muscles, and indeed any tissue it encounters. The mass is semi-elastic, and as the skin is approached and thinned, soft fluctuating areas may be detected. These break down and result in sinuses, which are often numerous and honeycomb the mass. The discharge is thin and sanious, or almost pure pus, and contains the globe-like bodies formed by the fungus. Pure pus and abscess formation is the exception, and is perhaps due to the presence of ordinary pyogenic organisms. The glands are not affected; there is no pain, and constitutional symptoms only occur when the seat of the disease is some part of vital importance. The parts first infected may heal completely, the disease spreading in some other direction; spon-

taneous cure has been observed. Acute cases are sometimes met with. The symptoms naturally depend upon the seat of the mischief; if the jaws be attacked, the process may extend to the neck, backwards to the pharynx and spine, or upwards to the skull. If the liver or internal organs suffer, characteristic symptoms, due to the lesion and interference with the functions of the organ, will appear.

Diagnosis.—From sarcoma the disease may be diagnosed by its very slow growth, painless course, the absence of secondary deposits in the glands, and by the presence of the fungus in the discharge and granulation mass.

When the liver or other internal organ is affected the diagnosis may be extremely difficult, as it may be impossible to secure the fungus.

Prognosis.—If the disease is, from its situation, not amenable to surgical treatment, it will prove fatal, but may persist for many months. Septic infection or some intercurrent mischief usually terminates the case.

Treatment.—Complete eradication of the granulomatous mass by excision or sharp-spooning, or both combined, is the only available treatment. Iodide of potassium internally is said to be useful.

MYCETOMA—MADURA FOOT

Etiology.—Mycetoma is due to a fungus closely allied to, and thought by Kanthack to be identical with, the actinomyces. Boyce and Surveyor regard it as distinct, and have cultivated from it a form of streptothrix; but as yet this has not been inoculated. Clinically there are many points of resemblance and contrast between actinomycosis and mycetoma. The latter is common among the natives of India who work in the fields bare-footed; inoculation takes place through a wound. The disease may attack the hands.

Signs.—Mycetoma is very chronic. It begins usually by the formation of one or more flat, raised, indurated tubercles or papules, which subsequently soften and break down, leaving an unhealthy sinus. Masses of granulation tissue develop in the connective tissue, and the bones, muscles, etc. are gradually invaded, the foot being much enlarged. The sinuses riddle the tissues and may freely intercommunicate. Abscesses may result. In the sinuses and granulomatous tissue, black deposits like coarse gunpowder.

or pale masses resembling fish-roe are seen ; these are the mycetoma colonies. Destruction is progressive and the disease may last for years ; it is unaccompanied by pain or constitutional symptoms. Deposits in internal organs do not occur.

Treatment.—The treatment is the same as that of actinomycosis. In advanced cases amputation should be performed.

CHAPTER VIII

Surgical Infective Diseases (*Continued*)

TUBERCLE

Tubercle is an infective inflammatory disease clinically characterised by its chronic, insidious course, by caseation and softening of the inflammatory products, and by its gradual invasion and destruction of the tissues unaccompanied by any attempt at organisation or perfect repair. Tubercle is, in almost all cases, a local disease, capable of eradication in situations permitting radical surgical treatment; but in some instances invasion of the body generally may occur and acute tuberculosis ensues. In rare cases, acute general tuberculosis may occur independently of any local tubercle. As compared with the extreme frequency of local tubercle, the general disease is very rare.

It is now generally admitted by pathologists that those conditions formerly called scrofulous or strumous are in reality tubercular. By the term scrofula is now meant a certain constitutional tendency to the development of low and intractable forms of chronic inflammation of a tubercular nature, as the result of the most trivial local causes. Scrofula is a term better abandoned, but long usage will probably ensure its survival for many years.

Scrofula or struma must not be regarded as a disease in the ordinary sense of the term, although it is true that the diminished vital resistance of the tissues, which is the leading characteristic of the condition, is a departure from the normal standard of health. Scrofula is, in fact, a condition predisposing to tubercular infection, but is not the disease itself. This predisposition doubtless lays the patient open to diseases of all kinds, and unfits him to battle against them; but the great majority, if not all, of the local lesions,

formerly described as scrofulous, are now known to be tubercular, although diligent search is often necessary for discovery, in the diseased tissues, of the *materies morbi* characteristic of tubercle.

Inoculation of susceptible animals with the inflammatory products from a so-called scrofulous or strumous lesion produces in them typical tuberculosis.

Etiology.—The scrofulous or tubercular tendency is usually inherited from the parents, but under certain conditions may be acquired. Hereditary transmission is not equally marked in all members of the same family, some of whom may entirely escape.

The parents are usually themselves tubercular, and if both suffer, transmission is more certain and severe; should one only be tubercular, it is worse if that one be the mother. Independently of the tubercular diathesis in the parents, any serious condition of ill-health, especially syphilis, may render the offspring tubercular.

The tubercular tendency may be aggravated or diminished according to whether the child, during its early years, be placed under conditions favourable to health or the reverse. Children living under bad hygienic conditions, especially in damp, overcrowded, and ill-ventilated dwellings, with their usual accompaniments of dirt and bad feeding, are specially prone to have their inborn tubercular tendency aggravated, or, should they have been born healthy, to acquire it. Dyspepsia is an important predisposing cause.

Tubercular children often betray the diathesis by their physical and mental attributes. There are two chief types—the sanguine and the phlegmatic.

The sanguine type is always hereditary, and is marked by mental acuteness, precocity, and vivacity. The child is usually tall, slender, and gracefully built, but the chest, especially in its antero-posterior measurement, is small. The features are delicately cut, the complexion good, and the child often very attractive.

The skin is delicate, clear, and fine, so that the subcutaneous veins are plainly seen. The hair is fair, fine, and silky; long downy hairs are often abundant over the back, shoulders, and forearms. The eye-lashes are long, abundant, and gracefully curved; the eyes large, bright, and intelligent-looking, and often blue in colour.

The phlegmatic type may be hereditary, but is often acquired, and is seen especially among the poor of large cities. It is closely allied to the rickety physiognomy. The child is ill-grown, stunted,

awkward, and ungainly. The mental condition is dull. The features are coarse, large, thick, and ugly. The skin is coarse and greasy, pale and pasty in appearance owing to the abundance of lymphatic tissue. The hair is coarse, and of a reddish, sandy, or dark brown tint. The finger ends may be clubbed and the nails curved.

It must not be supposed that all tubercular children will fall under one or other of these types, for there are many gradations between them. A mixture of the two is sometimes spoken of as "pretty struma."

While the strumous diathesis is a strong predisposing cause of tubercle, its presence is by no means necessary for infection. Generally speaking, man is not susceptible to tubercle; for it must be remembered that probably all of us run daily risk of infection. Tubercular affections occur especially in early life, and are comparatively rare after the age of twenty. Sometimes the disease manifests itself in later life, especially in women (senile tubercle).

The *tubercle bacillus* was discovered by Koch in 1882, and is a short, thin, slightly curved, non-motile organism, multiplying in the body by fission and spores. It is an aërobic obligate parasite (see p. 91), although capable of *living* outside the host. The organisms are sometimes very few in number, and difficult of detection. They are readily stained by Gram's method or by Ziehl's fuchsine stain; they are present in the pus and discharges from tubercular lesions, in the tissues, and especially in the epithelioid and giant cells to be presently described. The organisms are readily killed by boiling, by carbolic acid, or by mercury solution, but the spores are more resistant than are the mature bacilli.

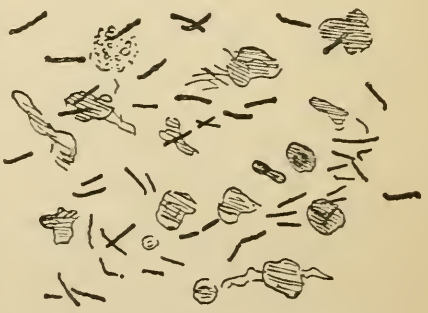

FIG. 31.—Tubercle bacilli in sputum.

Modes of infection.—The bacilli usually gain entrance through the mucous membrane of the throat or respiratory tract and show a marked preference for lymphatic structures. In many cases the entry of the bacillus is favoured by some antecedent inflammation or slight injury. The organism taken with the food is usually destroyed in the stomach, so that primary inoculation of the intestinal mucous membrane does not occur; but in cases of

phthisis, the large quantity of bacilli swallowed with the sputum may excite intestinal tuberculosis.

Tuberculous meat, unless thoroughly cooked, and the milk of cows with tubercular udders, may convey infection; such modes, however, ought never to occur if due care be taken by butchers and cowkeepers.

Very rarely a wound may be the seat of inoculation (see Anatomical Wart, p. 153).

Development and spread in the tissues.—The bacilli are taken up by the leucocytes, and multiplying in the tissues, lead to the formation of tubercle nodules. The disease may spread by local invasion of the tissues, or by the lymphatics to the nearest glands. In other cases dissemination through the blood-stream occurs. Each focus of tubercular mischief may be the source of further infection, the bacilli being taken up by leucocytes and carried by the lymph- or blood-stream to parts where they can develop. No doubt multiple tubercular lesions are due to embolic infection; and the occurrence of general tuberculosis is dependent partly on the dose of the poison, and partly on the marked predisposition of the tissues.

Morbid anatomy.—Naked-eye appearances.—Tubercular inflammation is characterised anatomically by the formation at the seat of inoculation of small nodules or miliary tubercles. These present somewhat different appearances according to their age. When first recognisable by the naked eye, the nodule, which is about the size of a pin's head or larger, projects somewhat from the surface, and is surrounded by a hyperæmic zone. It is dense in consistency, semi-translucent, and grayish in colour.

After a time, when caseation occurs, the consistency is lost and the nodule assumes a yellow colour and cheesy appearance (yellow or crude tubercle). By the coalescence of adjacent nodules a larger mass is formed (conglomerate tubercle), and if the disease be still active this goes on increasing in size. Round such a mass the tissues are studded with tubercles, each surrounded by a hyperæmic zone, and thus gradual invasion of the tissues takes place. The tubercular nodule is non-vascular—a fact of considerable importance in regard to its subsequent fate.

Microscopic anatomy.—When examined microscopically, a miliary tubercle as above described is seen to consist of numerous smaller nodules, each of which is constructed as follows. In the centre is a multi-nucleated giant cell with branched processes; these are continuous with a fine fibrillar reticulum which pervades

the microscopic tubercle, but which is not readily demonstrable (Fig. 33, p. 148). The existence of this reticulum is denied by some authorities. The nuclei are usually arranged at one pole of the giant cell, the bacilli at the other, as if there were negative chemiotaxis between them; in other cases the nuclei may lie round the margin, the bacilli then being centrally disposed. Sometimes two or three giant cells are present, one usually being more developed than the others. The origin of the giant cells is accounted for in different ways by different observers, and perhaps they do not always arise in the same manner.

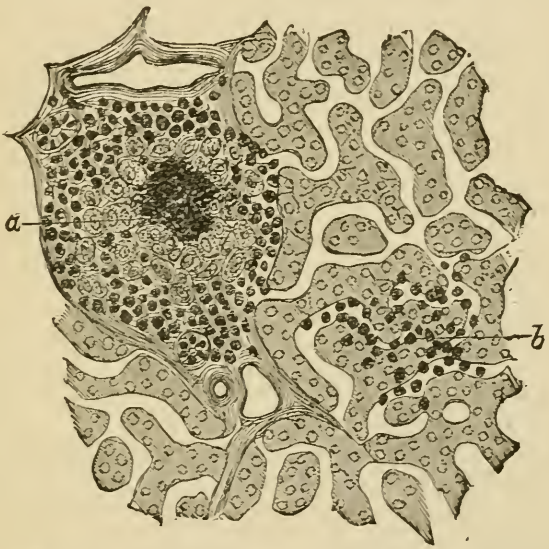

FIG. 32.—Miliary tuberculosis of the liver (Ziegler). *a*, mature tubercle; *b*, cluster of small cells forming an incipient tubercle.

According to Koch and Metchnikoff, they are aggregations of leucocytes (phagocytes); Baumgarten denies this and attributes them to the connective tissue or endothelial cells, which, he asserts, are large, because they have undergone incomplete division—the nuclei dividing, but the protoplasm merely increasing. Others consider that the giant cells are merely lymph coagula, the nuclei being cells caught up in the coagulum; but this would seem to be negatived by the peculiar and constant arrangement of the nuclei and bacilli.

The giant cell is surrounded by a zone of large mono-nuclear, epithelioid cells with granular protoplasm; these are derived from the endothelial and connective tissue cells. In and between these cells bacilli are present. Outside this is a zone of small round

cells (migrated leucocytes) gradually becoming less numerous as the healthy tissue is reached (Fig. 33).

Such is the anatomy of a typical nodule; but in some cases, especially if the formation has been rapid, the type is departed from and the giant cell may be absent.

The peculiar association and grouping of these cellular elements is characteristic of tubercle, but the individual cells themselves are not; thus giant cells are met with in developing granulation tissue,

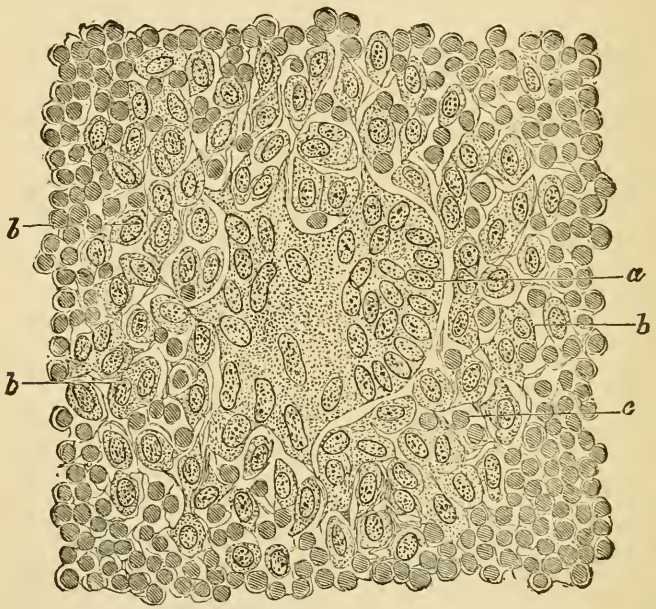

FIG. 33.—Tubercular nodule from a case of white swelling of the knee-joint. *a*, giant cell; *b*, epithelioid cells; *c*, lymphoid cells. (Ziegler.)

carious bone, and myeloid tumours, and the epithelioid and small round cells are commonly present in simple inflammations.

The distinctive characters of the tubercular giant cell lie in the arrangement of its nuclei and the presence of the bacilli.

Ultimate fate of the tubercular tissue.—Caseation is a constant occurrence in tubercular nodules. The change is due in part to the absence of vessels, in part to the irritating effect of the toxines produced by the bacilli. Caseation occurs first in the centre of the tubercle—that is, in the part most remote from the surrounding vessels and most favoured by the organism and its spores. The process is usually slow, but may be rapid. The giant cell becomes granular and loses its characteristic appearance, the nuclei and bacilli being no longer distinguishable; the

epithelioid and round-celled zones follow suit, and the whole is converted into a cheesy mass.

The caseous material possesses infective properties, and may excite suppuration in the neighbouring tissues; the pus, mixed with the softened caseous material, forms a tubercular abscess. Sometimes no pus is formed, but the caseous mass softens and is mixed with fluid derived from the surrounding tissues. A chronic tubercular abscess is surrounded and limited by a more or less dense capsule of new connective tissue; the wall is the seat of tubercular nodules with bacilli, by the successive breaking down of which the abscess continues to increase in size. The contents of such an abscess are not pure pus, but consist of sero-purulent fluid, floating in which are shreds and masses of curdy, caseous material.

Caseation does not, however, necessarily entail softening and abscess formation. The fluid may be absorbed and the caseous patch dry up and become encapsuled by new connective tissue. Such a mass is practically a foreign body, and is incapable of undergoing any further active change; but sometimes, even after many years, some trivial cause may reawaken the mischief, and thus an abscess forms (residual abscess). Caseous masses may become partially, but are rarely wholly, absorbed.

The amount of new scar tissue may be considerable, and may enclose a small caseous patch so that the bacilli are completely isolated and spontaneous cure results. A caseous mass may become calcified. According to Metchnikoff, the process of calcification is directly dependent upon the activity of the giant cells; he says, "The bacillus defends itself by the secretion of cuticular membranes, and probably also by the production of toxines, while the giant cell secretes a calcareous deposit, by means of which it walls in the bacillus and usually succeeds in killing it. The giant cell also probably produces digestive fluids which aid it in attacking and digesting the bacillus."

If repair occurs after abscess or ulceration due to tubercle, it is always more or less imperfect. The scar is thin and may break down again, especially if it is irritated. On account of the feebleness of the scar tissue, cicatrisation and contraction are but little marked.

Diagnosis.—The diagnosis of tubercle is usually easy and is rendered certain by the discovery of the bacillus. The general state of the patient, his age, the history of the case, the chronic nature of the mischief and its destructive tendencies, and finally, the seat of the disease are the main diagnostic features. The lesions

of the late stage of hereditary syphilis sometimes very closely resemble those of tubercle (see p. 206). In doubtful cases, *e.g.* renal tuberculosis, experimental inoculation of a susceptible but healthy animal, *e.g.* a guinea-pig, is a valuable means of diagnosis.

Prognosis.—The prognosis depends upon whether the seat and extent of the mischief admit of radical surgical treatment.

Generally speaking, it may be said that localised tubercle which can be entirely removed can be cured. If the disease be not amenable to such treatment, it usually runs a progressive course and terminates fatally. Spontaneous cure occasionally occurs when the disease is limited and the patient not very susceptible.

Principles of treatment in surgical tuberculosis.—General treatment.—Every endeavour must be made to maintain and improve the general health of those suffering from tubercular lesions. Warm clothing, good hygienic surroundings, with suitable and abundant food, are essential. The digestive and excretory functions must receive special attention, for unless these are acting properly, healthy nutrition is impossible. Fresh country air, preferably at the seaside, and in a dry and equable atmosphere, is most important; and the patient should enjoy as much exercise in the open as possible. It is now fully recognised that fresh air is one of the most important means of combating tubercular disease. Residence at some bracing seaside resort such as Margate, or in the higher Alpine regions of Davos or St. Moritz, and other well-known health resorts, is advisable in bad cases, but is unfortunately within reach of the few only. A sea-voyage to the Cape or Australia may be taken with advantage when the tubercular tendency is well marked, and after any local lesion has been cured or benefited by treatment. During winter months residence in Egypt is advisable. The open-air treatment of tuberculosis has been attended with marked benefit, about two-thirds of the patients showing great improvement. On the Continent this method has been largely introduced, and Sanatoria are numerous; the best known are those of Nordach in the Black Forest, Davos, and Falkenstein in the Taunus. All the rooms face south, and are protected from the prevailing wind; the patients spend from 9 A.M. to 10 P.M. in the open air, and sleep with the windows open. The diet is liberal, rest is enforced, and cold douching with massage, or the latter alone (according to the strength of the patient), are useful adjuncts to the treatment.

Tonics should be given in accordance with the requirements of the case: cod-liver oil, maltine, Fellow's syrup, and the phosphate

or iodide of iron are those usually employed; if there is marked anæmia, quinine, strychnia, and the astringent preparations of iron with vegetable bitters are indicated. The internal administration of guaiacol seems to have a powerful influence in arresting the progress of tuberculosis.

Local treatment.—Before softening has occurred, much may be done to favour the arrest of the tubercular mischief, unless the patient's susceptibility is very marked. In all cases the general principles of treatment are the same, but the details of their application necessarily vary with the seat of the disease. Rest of the diseased part and its surroundings is of primary importance; it should be complete and long-continued. Counter-irritation is sometimes productive of good results, and probably acts by drawing an increased quantity of blood to the part, hence bringing more phagocytes to deal with the *materies morbi*. Blistering and the application of the actual cautery may be useful in tubercular disease of bones and joints. The application of iodine to the skin over tubercular glands and abscesses is useless and frequently mischievous; it is the refuge of the destitute.

Any source of local irritation must be removed, especially in the case of the enlarged glands so often seen in tubercular children; in such cases removal of irritation about the scalp, throat, or mouth is often followed by marked improvement, with almost complete subsidence of the glandular swelling.

When caseation and softening have occurred, the only treatment of any avail lies in the complete eradication of the whole area of the disease by surgical operation, and the sooner this is done the better; for not only does delay allow of local extension, but each tubercular focus is capable of producing others, or of inducing general tuberculosis. If possible, the diseased mass should be dissected out like a tumour, but when circumstances will not permit of this, it must be eradicated by the sharp-spoon and scissors and the wound thoroughly dusted with iodoform, which seems to have some specific influence in tubercle.

The special treatment of tubercular disease of various organs and tissues will be referred to in their proper sections.

Tuberculin.—In 1890 Koch prepared a glycerine extract of pure cultures of the bacillus tuberculosis known as tuberculin. By experiment it was found that this substance, when injected beneath the skin, had a wonderful effect on tubercular lesions, and hopes were entertained that it would prove to be a powerful and effective antitoxin. These hopes were not, however, fulfilled,

and the drug proved to be dangerous, as it not infrequently wakened quiescent tubercle into fresh activity.

Early in 1897 Koch published (*Deut. Med. Woch.*) an account of a new antitoxin which he calls tuberculin R. This preparation is yet on its trial. In cases of lupus vulgaris the injections produce rapid and surprising improvement, but this appears to come to a standstill after a time, and cure cannot be said to result. The drug has also been used with benefit in other tubercular affections. It must be used most carefully and with full aseptic precautions; it often contains septic micro-organisms. The dose is gradually increased from one to twenty milligrammes, and an injection is made every second day.

THE SPECIAL SEATS OF TUBERCLE

No organ or tissue of the body is exempt from tubercular disease, but some are especially prone. In surgical practice the glands of the neck, the bones, joints, genito-urinary tract, peritoneum, skin, and subcutaneous tissue and mucous membranes are the usual seats of the disease. Of the viscera, the lungs are the common seat, but tubercle of the intestines, spleen, liver, brain, pia mater, and serous membranes is common. The disease, as it attacks the various parts, will be fully described in the chapters relating to them, but tubercle of the skin and subcutaneous tissue will be shortly described here.

Subcutaneous tubercular abscess unconnected with disease of a bone, joint, or glands results from a deposit of tubercle in the subcutaneous tissue. The abscess is very chronic and not accompanied by the usual signs of inflammation. It is localised and elastic, but when softening occurs, fluctuation becomes more or less evident. The overlying skin is gradually undermined and thinned, becomes red and slightly tender, and when it gives way the contents are evacuated and an unhealthy suppurating wound is left. This may continue to discharge and perhaps burrow in various directions through extension of the tubercular process, and by the complete destruction of the skin an unhealthy ulcer may result. Such abscesses require the treatment given at p. 49.

Tubercular ulcers may be met with on the skin or mucous membranes. Ulcers in the latter situation are met with (*q.v.*) in the tongue, intestine, nose, larynx, anus, etc. The skin ulcers are chronic and not amenable to ordinary treatment, but quickly heal if thoroughly sharp-spooned. The base is dirty and may be studded

with granulations; the edges are ragged, irregular, undermined, and often livid. The base and edges may be indurated from the presence of tuberculous material, to the breaking down of which increase in size of the ulcer is due. Treatment consists in free removal of the diseased structures by the sharp-spoon and the application to the raw surface of chloride of zinc paste or iodoform, followed by some simple unirritating dressing, such as boracic ointment. The resulting scars are often depressed. Small ulcers should be completely excised; the edges of the skin are then united, or the gap, if too large for this, is covered by grafts according to Thiersch's method.

Anatomical or butcher's wart.—Those engaged in *post-mortem* examinations or in slaughter-houses occasionally develop on the hands, especially over the knuckles, a persistent warty condition which shows little or no tendency to ulcerate, although it is very resistant to treatment. The absence of ulceration is in marked contrast to what occurs in lupus, although these conditions are certainly allied. The tubercle bacillus can often be found in anatomical wart, but it is by no means certain that all cases are tubercular.

Treatment consists in the application of nitric acid or some strong caustic, and if this fails, the warty patches must be freely sharp-spooned.

Lupus.—Lupus vulgaris is a disease of the skin and mucous membranes due to the presence of the B. tuberculosis. Fully formed tubercular nodules are rarely met with, the morbid material consisting chiefly of round cells; the bacilli are demonstrated with difficulty. Lupus is usually met with in girls about the age of puberty, and sometimes in much younger patients; it may also occur about the hands of elderly people.

The favourite seat of the disease is the nose and adjacent parts of the face. It begins in the form of raised reddish or brownish papules, which contain a material not unlike apple jelly; these sooner or later break down and lead to ulceration. The adjacent patches and ulcers coalesce and produce considerable tracts of disease round which isolated nodules of varying size are readily distinguishable. The broken-down patches are covered with a loosely adherent unhealthy scab, beneath which pus is imprisoned. As the disease advances, the mucous membrane of the nose may become affected; the alæ nasi may be destroyed and the cartilages and bones necrose, and hence the most unsightly deformity results. Sometimes the disease spreads to the mucous membrane of the

lips, mouth, and palate, and patches of it may be present in various parts of the body. The glands of the neck are not infrequently enlarged, and the patient is pale, anæmic, ill-developed, and unhealthy.

Treatment.—The ideal treatment is excision of the affected portion of skin, followed by grafting the raw surface by Thiersch's method. Healing is rapid, and the resulting scar is sound and good. In many cases, however, the disease has extended so widely that this plan is impracticable, and under such circumstances it must be eradicated by sharp-spooning. The patient is anæsthetised and every particle of diseased and softened tissue is scraped away; the raw surface is then treated with chloride of zinc paste well rubbed in by means of a small piece of wood or a glass rod. The surface is best dressed with a little iodo-vaseline spread on thin butter-cloth. The case must be kept under supervision, and at the first sign of a recurrence of the disease the sharp-spoon must be at once resorted to. The employment of tuberculin R. is referred to at p. 152.

The general treatment is that applicable to tubercle.

LUPUS ERYTHEMATOSUS

Although this is not a tubercular affection it is convenient to describe it here.

The disease usually occurs in women during adult life.

Seat and morbid anatomy.—Lupus erythematosus may affect any part of the body, but shows a special predilection for the neighbourhood of the nose and cheeks. The disease is characterised by enlargement of the small veins, by congestion and round-celled infiltration of the cutis, with considerable increase in the sebaceous secretion. Hyperæmic plaques appear on the surface, and after a time become covered by scabs formed of desquamated epithelium and sebaceous matter. By coalescence of the plaques a large area of disease results which spreads over the cheeks in a remarkably symmetrical manner, the affected area taking much the shape of a butterfly's wings. In the course of time the invasion of the round cells leads to atrophy of the normal tissues, and a thin white scar remains. The disease is very liable to relapse, and may persist for a very long time. There are practically no subjective sensations beyond a little itching, and the general health remains good. Ulceration does not occur.

Treatment.—Linear scarification is one of the best forms of

local treatment, it obliterates the vessels and promotes absorption of the round-celled infiltrate; the cuts should be about an eighth of an inch apart over the whole area, and may be crossed by others at right angles to them. The application of mercurial ointment, or of a 2 per cent solution of resorcin, 10 per cent pyrogallic acid ointment, or tincture of iodine are all to be recommended, and should be used if scarification is declined by the patient.

CHAPTER IX

Surgical Infective Diseases (*Continued*)

The Venereal Diseases[1]

GONORRHŒA

Gonorrhœa is an acute infective inflammation of the urethra or vaginal mucous membrane, sometimes accompanied by complications due to local invasion, to auto-inoculation of the mucous membranes, or to invasion of the lymphatics or blood. One attack is not protective.

Etiology.—The gonococcus (Fig. 34) is the infective agent. It occurs in pairs or groups of four or its multiples, and is present in the pus, the detached epithelium scales, and in the intercellular substance. It is somewhat reniform, the concave margins being opposed. It is cultivated with great difficulty, and stains well with methyl-violet, but not by Gram's method. The organisms are less numerous when the disease has been present some time. A similar organism is sometimes present in the healthy urethra, and in cases of gonorrhœa the ordinary pyogenic cocci are associated with the gonococcus. It is probable that the pathological effects of gonorrhœa and its complications are due to mixed infection with the gonococcus and pyogenic microbes.

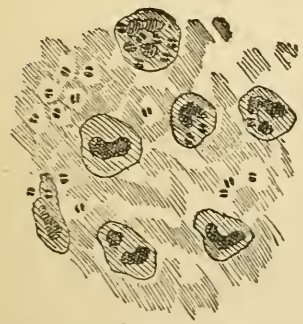

Fig. 34. — Gonococci and pus cells. (Drawn by G. Coltart.)

Some people are more susceptible to the disease than are others,

[1] It has been considered more convenient to group these diseases together, although gonorrhœa and chancroid may be regarded as local diseases, and syphilis as general.

and suffer more severely. A long and tight foreskin, hypospadias, and uncleanliness favour infection. The tubercular, rheumatic, and gouty are liable to suffer for a long time, and to be affected by certain complications. As a rule, the first attack of gonorrhœa is the most severe.

Morbid anatomy.—In men the urethra, especially at the fossa navicularis and bulb, is the seat of the disease, the glans and prepuce often participating. In women the vagina and vulva are primarily affected, but extension to the urethra is common.

The organisms invade the epithelium cells, pass between them into the deeper structures, and may then spread by the lymphatics. They excite acute inflammation; the mucous membrane acquires a bright red colour, and is swollen and highly sensitive. The epithelial cells proliferate, and some of them becoming detached, are carried away by the copious purulent discharge which soon occurs. Sometimes flakes of lymph cover the inflamed surface, and are washed away by the urine, or the process may be so intense that phlyctenular ulcers and erosions occur, which may be the seat of subsequent stricture or warty growths. The inflammation gradually extends backwards, but is often arrested at the bulb or membranous urethra; extension to the lacunæ and ducts opening into the urethra may lead to perineal abscess, prostatis, epididymitis, or vesiculitis; if the erectile tissue of the corpus spongiosum is involved, its distensibility is diminished by bands of lymph, and erection is consequently imperfect and painful (chordee). Extension to the bladder and upper urinary tract is rare. Involvement of the lymphatics may occasion lymphangitis and bubo, or cellulitis in women. Gonorrhœal rheumatism and pyæmia are due to dissemination of the poison by the blood-stream, through involvement of the prostatic plexus, more rarely through the dorsal vein. The various complications thus indicated do not occur with equal frequency, and they will be considered in due course. In women similar invasion is noticeable, but different organs are necessarily involved. The cervical canal and body of the uterus, the Fallopian tubes, ovaries, and pelvic cellular tissue may all suffer, and doubtless many cases of chronic tubal disease and ovaritis are due to antecedent gonorrhœa.

SIGNS AND SYMPTOMS OF GONORRHŒA IN THE MALE

The incubative stage is usually about four or five days, but may be longer or shorter. In the majority of cases the shorter the

incubation the more severe the course of the disease. During this period the patient may experience some frequency of micturition with slight scalding, accompanied by swelling and itching at the meatus.

When the acute stage comes on, the itching, redness, and swelling of the meatus are more marked, and the whole penis may be slightly swollen, congested, tender, and hot, especially along the corpus spongiosum. Micturition is frequent, and accompanied by smarting and burning pain, which is sometimes very severe. For the first few hours the discharge is scanty and watery, but soon becomes copious, thick, and creamy, and of a greenish-yellow colour. The urine is turbid from admixture of discharge, and shreds of lymph are seen floating in it. Sometimes blood, proceeding from an erosion, may escape with the discharge; or hæmorrhage may be more severe and constant owing to rupture of an engorged vessel during erection. Painful and persistent erections with a curved condition of the penis are common at night; they are due to the genital irritation and the curvature to the incomplete distension of the erectile tissue already mentioned. As the inflammation extends backwards the symptoms are aggravated, especially if the prostatic urethra and neck of the bladder are involved. In such cases the frequent and painful attempts at micturition—only a few drops of urine being voided at each attempt—may be very distressing and alarming to the patient, who is fearful of complete retention and stricture. The congestion of the mucous membrane, swelling of the veru-montanum, and associated spasm of the peri-urethral muscles may cause retention. The dorsal lymphatics are often enlarged and tender, and the superficial inguinal glands may participate, but rarely suppurate. In some cases, small, nodular and tender swellings may be present along the urethral canal; these are due to inflammation of the lacunæ, and may be the starting-point of peri-urethral abscess. Usually gonorrhœa is unaccompanied by general disturbance; but sometimes the general health suffers, especially in weakly persons, or if pain and chordee occasion sleepless nights. Slight fever is sometimes present.

The acute stage passes off in from one to four weeks, according to the severity of the attack. The local signs subside, and the discharge becomes less copious and more watery; it may disappear in another ten days or so, or remain indefinitely as a gleet, appearing in the early morning when the urethra has not been washed out by the urine for some hours.

Prognosis.—Gonorrhœa runs its course in about a month or six weeks, but if the patient is in feeble health, or neglects the treatment prescribed, it may last much longer, and be followed by a chronic gleet. A narrow meatus, stricture, previous attacks, and chronic alcoholism are unfavourable to speedy cure. The subject of gleet is considered under Diseases of the Urethra, vol. iii.

Treatment.—General.—At the onset of the disease the patient should keep as quiet as possible, and if the attack be very severe he may with advantage remain in bed. Horse- or bicycle-riding and all forms of violent exercise must be prohibited. The bowels should be freely acted on and regulated by a saline aperient. The diet must be light, plain, and unstimulating; malt liquors, alcohol, coffee, tea, sauces, and highly-seasoned dishes being interdicted. Milk, barley-water, soda-water, and the like should be freely taken. If the smarting during micturition is severe, it may be relieved by the use of the carbonate or citrate of potash with tincture of hyoscyamus, by hot baths, or belladonna suppositories; and if the pain be extreme, 10 minims of a 5 per cent solution of cocaine may be injected into the urethra before micturition.

During the acute stage much relief is obtained by the use of the hot bath night and morning, the patient remaining in it from ten to twenty minutes. Troublesome chordee should be treated by the administration of bromide of potassium and chloral hydrate, or by the use of the hot bath before the patient retires to rest, followed by a suppository of three grains of camphor with opium or belladonna. The painful erection is immediately relieved by immersing the penis in cold water.

Strict personal cleanliness must be observed, and an antiseptic gonorrhœa-bag should be worn to prevent staining of the linen by discharge. The practice of placing a piece of lint or cotton over the end of the penis and beneath the foreskin for this purpose is to be condemned. A suspender should be worn. The patient should always be warned of the contagious nature of his discharge, especially with regard to accidental inoculation of the conjunctiva.

The **special treatment** consists in the administration of certain drugs internally and topical applications by urethral injection, the latter being the most important. Internally copaiba, cubebs, and sandal oil are the drugs of greatest value, though they are uncertain in their action, and liable to cause gastric disturbance and dyspepsia. They are excreted by the kidneys, and have apparently a direct effect upon the urethra. These drugs are specially useful when the acute stage is subsiding, and are contra-indicated during that stage. Copaiba

has the disadvantage of sometimes producing an extensive irritable urticarial rash, perhaps with associated constitutional symptoms; occasionally cubebs acts in a similar manner.

Copaiba is best given in capsules on account of its nauseous taste; a small dose is given at first, for fear of inducing the toxic symptoms referred to, but the dose may be gradually increased up to 30 minims or a drachm thrice daily.

Cubebs is more likely to upset the digestion than is copaiba; it is best administered as the fresh-ground powder, one teaspoonful in a little warm milk being taken three times a day shortly after meals.

Sandal oil should be given in 10-20 minim doses, contained in capsules, three times a day. It is less irritating and more easily borne, but also more uncertain in its action, than copaiba or cubebs. All these drugs must be persevered with and taken for some time before being discarded as useless.

Turpentine, Canada balsam, buchu, uva ursi, quinine, and iron are sometimes useful if copaiba, cubebs, or sandal oil cannot be tolerated.

Injections.—During the acute stage, when there is much congestion, copious discharge, and severe smarting, the urethra should be cleansed three or four times a day by the injection of tepid water. At this period astringent lotions are ill borne, and may materially aggravate the condition, besides being very painful. The permanganate of zinc, gr. $\frac{1}{10}$ to an ounce of distilled water, is very mild and most useful; it may be used after the first few days.

As the acute stage subsides astringent and antiseptic injections are indicated. It will often be found that an injection suiting one case is unsuitable for another. The chloride of zinc, gr. $\frac{1}{6}$, ext. belladonna, gr. 2 to an ounce of distilled water, is a useful injection in most cases. Sulpho-carbolate of zinc, gr. 1-2; sulphate of zinc, gr. 1-3; tannic acid, gr. 1; nitrate of silver, gr. $\frac{1}{4}$-$\frac{1}{2}$, are all useful, and may be combined with extract of belladonna or acetate of lead, gr. 1-2, ad ℥i. Mercuric chloride, 1 : 5000, is recommended by some, but is often productive of more harm than good, as it is liable to cause considerable irritation. Certain preparations of silver are now much in vogue, especially on the Continent. Argentamin, largine, itrol, argonin, and protargol are the preparations employed, the last, introduced by Neisser, being the most favoured because of its unirritating properties and more beneficial action as a bactericide. Protargol contains about 8 per cent silver nitrate, and should be used as a 1 per cent solution. The injections are

made thrice daily, being retained the first time about five minutes, the second fifteen, and the third thirty. The treatment must be kept up for about a month, but lessens the severity of the disease and prevents its extension backwards. The advantages claimed for protargol are borne out by experience, although some cases prove refractory. In chronic cases affecting the deep parts of the urethra, a 20-25 per cent solution may be applied topically through the endoscopic tube or by Guyon's injector.

Largine ($\frac{1}{4}$-$\frac{1}{2}$ per cent solution) is said to be as useful as protargol, if used in the same way. In obstinate cases the injection of the four sulphates (alum sulph., zinc sulph., ferri sulph., āā gr. 20-30; cup sulph., gr. 2, ad ℥viii.) may be tried.

The use of injections is contra-indicated if there is much local congestion and irritation, or if they aggravate the local condition and cause much pain. If tolerated, their strength may be slightly increased about every four or five days, but the use of very strong solutions is to be avoided. When the membrano-prostatic portion of the urethra is affected, it is generally necessary to apply the lotions topically through the endoscopic tube.

Method of using an injection.—Injection should be made from three to six times a day, but is best avoided the last thing at night. Previous to injecting, the patient should empty the bladder, and then wash out the urethra with a syringeful of tepid water. About two drachms of the lotion (slightly warmed) is then carefully injected, and retained for about two minutes or longer by holding the glans penis. The test of a successful injection is that when the fluid is allowed to escape it does so in a jet, and does not merely dribble away.

Irrigation method.—Janet, who introduced this method, claims that it is superior to any other and may cut short the disease in from five to seven days. This plan has not received the attention in the country which it merits; it has the disadvantage of being troublesome to carry out. Janet recommends the use of permanganate of potash solution, 1 : 4000 to 1 : 1000; protargol 1 per cent, and itrol 1 : 4000, are also highly spoken of by some. The irrigation is performed twice daily, with the weaker solutions to begin with, and in three or four days only once in twenty-four hours with a stronger solution, after another ten days irrigation is carried out once in forty-eight hours, and the permanganate solution is of the strength 1 : 1000. The fluid is introduced by gravity (the vessel holding it being about 20-40 inches above the penis, according to the length of urethra involved); a glass cannula is held

within the canal, and about one to two pints is used at each irrigation. If congestion of the urethra is caused, the strength of the solution must be diminished. The test of the cure is in the absence of gonococci from the discharge.

Medicated bougies are much praised by some surgeons, but often produce irritation without any proportionate good. The bougies, composed of iodoform, gr. 5, eucalyptus oil, ♏ 10, and cocoa butter, are well oiled and introduced at night, a gonorrhœa-bag being worn to prevent soiling of the linen. A bougie may be passed each night for a week, and during the day a mild astringent injection should be used.

The abortive treatment of gonorrhœa is not to be recommended. It consists essentially in the application through the endoscopic tube of a strong solution of nitrate of silver to the inflamed area, with the internal administration of large doses of the resins. This treatment is attended by considerable risk of severe inflammation.

SIGNS AND SYMPTOMS OF GONORRHŒA IN THE FEMALE

The entrance of the vagina and the vulva are the parts of primary attack, the disease spreading upwards towards the uterus, and very often to the urethra. In women, gonorrhœa may remain undiscovered for some time, since there is often no pain or smarting on micturition, and the discharge and itching induced by it are attributed to leucorrhœa. When the vulva, and especially the nymphæ, are inflamed, the patient's attention is quickly aroused by the smarting pain on micturition, and by the swelling and irritation about the parts, coupled with the profuse greenish-yellow discharge. On examination the nymphæ will be found swollen and projecting, intensely red, and perhaps excoriated. The affected mucous membrane is of a bright red colour, congested, swollen, and exquisitely tender, so that a thorough examination is difficult. Phlyctenular ulcers and erosions may be present in bad cases. The discharge is very irritating, often highly offensive, and comes from the vaginal mucous membrane and not from the cervix uteri unless this be involved. If the urethra is affected, discharge can be squeezed out of it by drawing the finger along it from above down; it may also escape from the orifices of Skene's ducts, which lie close to the meatus. A purulent discharge from the urethra is practically diagnostic of gonorrhœa, as inflammation of other origin does not affect this canal. The acute stage usually subsides

under treatment in from ten to fourteen days. If the disease becomes chronic, the upper part of the vagina and the cervix are chiefly affected.

Diagnosis.—Leucorrhœa may be distinguished from gonorrhœa by the absence of acute inflammation, and by the facts that the discharge comes from the cervix uteri and not from the vagina, and that the urethra is not involved. If gonorrhœa spreads to the cervical canal, there will, in addition to the vaginal discharge, be a purulent discharge from it. In all doubtful cases the organism should be sought for with a view to clearing the patient's reputation, or in view of future legal contingencies.

Prognosis.—If the case comes under treatment early the prognosis is good, as the affected parts are easy of local treatment; but if it has been neglected and become chronic, the disease is often very obstinate, and may lead to grave complications to be presently mentioned.

Treatment.—Topical applications may be applied by means of tampons, which may be retained. They are practically the same as those recommended for injection in the case of the male.

The employment of rest, hot baths, and free douching of the vagina with boracic acid (gr. 4, ad ℥i.), alum (gr. 5, ad ℥i.), or acetate of lead (gr. 2, ad ℥i.) will speedily allay the acute symptoms. Severe pain may necessitate the local application of a 5 per cent solution of cocaine. Strict personal cleanliness must be observed; the nymphæ should be separated by a piece of lint, and a sanitary towel worn. If the urethra is affected, balsamic remedies internally and injections locally are indicated.

When the acute symptoms have subsided, any local inflammation and patches of erosion may be treated through the speculum by solutions of nitrate of silver, glycerine of tannin, or zinc salts. They may be used of greater strength than advised for urethral injection. When gonorrhœa affects, as it so frequently does, the cervical canal, great care must be taken that this receives adequate treatment, or the persistence of the disease may lead to serious after-consequences.

COMPLICATIONS AND SEQUELÆ OF GONORRHŒA

The various complications do not occur with equal frequency, and some people are more liable to suffer than others. Neglect of due care during treatment favours their occurrence.

Complications common to the sexes.—**Gonorrhœal ophthalmia.**—Infection of the conjunctiva may be due to auto-

inoculation, but occasionally surgeons are inoculated after examining a gonorrhœal case. The patient should always be warned of this danger. The disease, which runs a rapid and destructive course, may result in loss of the eye unless prompt treatment be adopted, and is accompanied by considerable mental and physical depression. Within a few hours of inoculation there is smarting pain in the eye, with acute congestion of the conjunctiva, which is much swollen and chemosed. Very soon there is copious purulent discharge, distending the swollen lids and dripping from the palpebral fissure. Pain in the eye and round the orbit is severe. Unless promptly cut short the cornea is implicated, suppuration and sloughing ensue, and the entire globe may be lost. Iritis and sclerotitis are sometimes met with in association with gonorrhœal rheumatism.

Treatment.—The other eye must be covered with a watch-glass let into a piece of strapping, which is fastened along the brow and nose. Under cocaine or a general anæsthetic the lids must be separated, the discharge washed away with boric acid solution, and the infected surface brushed over with a solution of silver nitrate (gr. 20, ad ℥i.); in a few minutes the superfluous solution should be washed away with water. This must be done once or twice daily, and in addition the eye should be frequently cleansed with boric acid or alum lotion. Protargol is recommended by Fürst as being less irritating and more reliable than silver nitrate. Pain may be relieved by the ice-bag, or morphia may be given. The general treatment must be of a tonic and stimulating character, in spite of the urethral discharge. If the eye sloughs the treatment for panophthalmitis must be employed.

Pyæmia is rare, and may spread from infective inflammation of the vaginal or prostatic veins. It runs the ordinary course of pyæmia from other causes, and is probably due to mixed infection.

Gonorrhœal or urethral rheumatism.—In the great majority of cases of urethral rheumatism gonorrhœa is the exciting cause, but it may arise from urethral irritation due to other causes, *e.g.* instrumentation. Fournier mentions a case in which the act of coitus was always followed by rheumatism the next day, independently of any infection.

It is much more common in men than women, and especially in those of a rheumatic or gouty tendency. Exposure to cold, chronic alcoholism, and previous attacks are causative agents. When once urethral rheumatism has occurred it is very likely to reappear with

any fresh irritation or gonorrhœal infection, and relapses are very common. Emery has recorded the case of a man who had nineteen attacks, with ten attacks of gonorrhœa.

Pathology and morbid anatomy.—It is probable that all cases of urethral rheumatism are not due to the same pathological change. Those of gonorrhœal origin are probably pyæmic, and the gonococcus has been found in the synovial effusion. Some think that the central nervous system, especially the medulla, is affected reflexly; whilst others, without much apparent reason, consider these cases to be of the nature of simple rheumatism.

The disease affects the larger joints, large fascial planes, peri-articular structures and ligaments, and occasionally the large nerve trunks, the sclerotic coat of the eye, etc., but very rarely the heart. The inflammatory change in the joints is essentially chronic, although marked by an acute onset; it attacks the knee joint more often than any other, although many may suffer. After repeated attacks the small joints of the fingers are sometimes permanently deformed, as in rheumatoid arthritis.

The peri-articular structures are infiltrated, and œdema often extends some distance beyond the joint; the synovial membrane becomes thickened, and there is an increase in the amount of fluid. Suppuration is very rare; more usually the disease becomes chronic, the fluid is gradually absorbed and repair ensues. Fibrous adhesion causing more or less complete ankylosis is occasionally found and is especially likely to occur after repeated attacks. Tendon sheaths and bursæ may be similarly affected, and cases have been recorded in which disseminated muscular atrophy was present (Gaston).

Signs.—Gonorrhœal rheumatism does not usually come on until after the third week of the disease, when the inflammation has extended backwards to the membrano-prostatic urethra. It may, however, occur within a few days, especially in recurrent attacks. At the onset the urethral discharge usually abates considerably and may temporarily disappear. The patient feels ill and depressed, and there is more or less fever, with general malaise and pains about the body and limbs; yet sometimes there is practically no constitutional disturbance. Occasionally pain in the affected joints is the only symptom; more usually this is accompanied by hydarthrosis which may be very persistent, partially clearing up at times only to relapse. In severer cases the peri-articular and ligamentous structures are involved, occasioning much pain, swelling, and constitutional disturbance, and perhaps resulting in

permanent stiffness. If more than one joint is involved, the disease is more serious and the patient's constitutional condition proportionately grave. The ankles, wrists, and elbows are often affected, the temporo-maxillary and sterno-clavicular articulations not infrequently, and no joint is exempt.

Severe pain may be experienced in the plantar or palmar fascia, along the ilio-tibial band or large aponeuroses. The sciatic nerve and sometimes other large trunks may be the seat of more or less severe neuralgia.

The disease is sometimes complicated by sclerotitis and iritis.

Diagnosis.—The presence of a urethral discharge, or in default of this, the detection of shreds in the urine; the obstinacy of the joint affection and the limitation of the disease to one or two joints, with perhaps implication of the nerves and fasciæ, and the slight constitutional disturbance, will usually lead to a correct diagnosis. The ordinary signs of acute rheumatism are absent.

Prognosis.—If the patient is young and has previously enjoyed good health, the prognosis is good, but the disease is often very chronic, may last as long as six months, and usually does so for as many weeks. Previous attacks are unfavourable not only to the duration of the disease, but to the complete recovery of the affected joints. Suppuration is very rare, fibrous ankylosis the exception, and resolution the rule. Those broken down in general health and with a marked gouty or rheumatic tendency are liable to suffer severely.

Treatment.—The primary indication is to cure the urethral disease, for so long as this remains, gonorrhœal rheumatism will prove intractable.

As regards the rheumatism, the patient must remain in bed and the affected joints be kept at perfect rest by the application of splints or other means suitable to the articulation. If the inflammation is very acute and accompanied by much pain and effusion, hot fomentations or repeated blisters should be applied, or the fluid may be removed by aspiration. If the course be more chronic, strapping with Scott's ointment is the best treatment, to be followed, when all active symptoms have subsided, by massage, friction, and gentle passive movement. The bowels should be acted on by saline aperients, and the secretion of urine favoured by diuretics. Salicylates are usually of no avail. The most useful drugs are iron, quinine, and iodide of potassium, combined with cod-liver oil and tonics.

The diet must be light but generous, and in spite of the

urethral mischief, a little wine is usually advisable in the depressed state of the patient.

When the patient is able to be moved without fear of a relapse he may advantageously go to Buxton, Bath, or Droitwich for further treatment, or may take a sea voyage.

Cystitis, pyelitis, and nephritis are rare complications, and are dangerous owing to the infective nature of the mischief.

Sympathetic bubo.—Sometimes the inguinal glands are slightly enlarged and tender, but suppuration is rare. Should it occur, the treatment is the same as for bubo from any other cause. It is more common in men than women.

Warts of the prepuce and glans penis or of the vulva are frequently, though by no means always, due to gonorrhœa. In the male, urethral warts may form at the site of an erosion.

Complications peculiar to the male sex.—Balanitis and balano-posthitis.—Inflammation of the prepuce and glans penis may occur, especially if the former is tight and redundant.

Inflammatory phimosis or paraphimosis.—These complications are very common in the uncleanly, especially if they have long and tight foreskins.

Peri-urethral abscess is usually due to the extension of inflammation along one of the lacunæ. The gonococcus finds its way into the peri-urethral tissue and abscess results. It is not a common complication.

Inflammation of the neck of the bladder does not usually come on until fourteen days or later from the time of infection. It is always associated with inflammation of the prostatic urethra. This condition is often incorrectly spoken of as "cystitis." The leading symptoms are frequent micturition with much pain and strangury; a little blood may be passed, and the urine is often turbid from admixture of discharge. The treatment consists in the administration of salines and alkalis with hyoscyamus, the use of the hot hip-bath, and of morphia or belladonna suppositories.

Prostatitis, sometimes resulting in acute abscess, may occur from extension along the ducts. In other cases the prostate is chronically congested, slightly enlarged, and tender, giving rise to persistent gleet. Prostatic involvement does not usually occur before the third week.

Epididymitis is one of the commonest complications of gonorrhœa, and occurs at the end of or later than the third week. It is due to direct extension along the vas deferens, and is usually unilateral, but may affect both sides.

Inflammation of the vesiculæ seminales sometimes occurs.

Hæmorrhage from the urethra may occur from a granular patch and is then slight in amount. When more severe and persistent it may come from rupture of a congested vein during chordee. In the latter case the bleeding soon ceases if the patient keeps quiet and bathes the penis in cold water; should it continue, an injection of tincture of hamamelis (℥ 20 ad ℥i.) may be necessary.

Congestive and spasmodic retention of urine is sometimes occasioned when the inflammation is very acute and affects the membrano-prostatic urethra. An organic stricture may also become temporarily occluded through congestion. The patient should be placed in a hot bath and encouraged to pass his urine in it; the bowels should be acted on, and rest and morphia suppositories with repeated baths usually relieve the trouble in a few hours. In bad cases a soft india-rubber catheter should be carefully passed.

Gleet and organic stricture may follow acute gonorrhœa.

Complications peculiar to the female sex.—Abscess of the glands of Bartholin is not uncommon.

Pelvic cellulitis may occur, especially in cases accompanied by ulceration.

Involvement of the uterus, etc.—Gonorrhœa may spread upwards to the cervix or body of the uterus, to the Fallopian tubes leading to pyosalpinx, to the ovaries and broad ligaments, and occasionally to the peritoneum. These complications are necessarily serious and frequently lead to chronic trouble.

SYPHILIS

Definition.—Syphilis is a chronic generalised infective disease with prolonged stages, manifesting its action by setting up peculiar inflammatory changes in various parts of the body, more especially the cutis, but also other more deeply placed connective tissue structures, so that every organ and tissue is liable to be attacked at some period during the evolution of the disease.

Syphilis presents itself in two forms—(1) the acquired, in which inoculation has occurred any time after birth; and (2) the hereditary, in which the fœtus has become infected *in utero*. These two forms will be separately considered, since the hereditary disease presents important differences in its course.

Etiology.—The poison of syphilis can only be communicated by contagion, and not by infection through the air. The contagion may also be transmitted to the offspring (heredo-contagion). That

the poison is a microparasite there can be little doubt; but, although Lustgarten and many other observers have described organisms which they claim to be the *contagium vivum* of syphilis, it must, for the present, be admitted that the evidence in support of these claims is inconclusive. Syphilis is peculiar to man. As yet the disease has not been recognised in the lower animals; and although it is true that Disse and Taguelic have reported the effects of inoculating rabbits, sheep, and dogs with the cultures of a micrococcus found by them in syphilitic sores, which produced chronic interstitial inflammation of the lungs and liver, fatty changes in the vessels, and granulomatous growths similar to those met with in syphilis, yet there was no definite proof that the animals had been rendered syphilitic. Whatever the poison may eventually prove to be, it is contained in the secretions from the primary and secondary lesions, and in the blood, but is not present during the tertiary period. It is practically certain that the normal physiological secretions do not contain the poison, and therefore can only convey the disease when they are mixed with inoculable discharge from a syphilitic lesion, *e.g.* the saliva when the throat or mouth is affected.

Syphilis has no relation to any other venereal disease, although it was formerly held by some that all such were simply different clinical manifestations of one and the same poison. Hutchinson considers that many of the non-syphilitic sores are really abortive manifestations of that disease. It is to be remembered that an ordinary soft chancre may also be inoculated with the syphilitic poison (mixed chancre).

As with other infective diseases, so with syphilis, some people prove more or less resistant to the poison, while others, especially such as are the subjects of chronic alcoholism, renal disease, tuberculosis, and similar conditions which diminish the general resisting powers of the body, suffer severely, and the disease may run a very rapid course (malignant and galloping syphilis).

ACQUIRED SYPHILIS

Modes of contagion.—The poison may be conveyed by direct or mediate inoculation through an abrasion or wound, but has probably no injurious effect when it is brought in contact with an uninjured surface.

Direct inoculation is the most common, and usually occurs on the genital organs during sexual intercourse; but the poison may also be communicated by the fingers, by kissing, by the inocula-

tion of a child's lip by a syphilitic nipple, or by unnatural sexual acts.

Indirect or mediate inoculation may occur through the medium of spoons, cups, pipes, etc., which have been used by a patient with secondary lesions in the mouth. The Eustachian catheter, probes, sponges, and surgical instruments must also sometimes be held responsible. In rare instances, chiefly on the Continent, vaccination has been the means of contagion, and thus great numbers in a community have been affected. Vaccine lymph does not contain the poison, and can only convey syphilis when it is mixed with blood; hence such lymph should never be used, even when the donor is apparently healthy. Doubtless many cases of syphilis in infants, which have been attributed to vaccination, are really instances of the hereditary disease, which has only declared itself *after* vaccination. Skin-grafting has also conveyed infection, and therefore grafts should always be taken from the patient needing them and not from an intermediary.

Symptomatology.—Syphilis bears a close analogy to the specific exanthems, but differs from them in its very prolonged course with intervals of apparent restoration to complete health, in the possible universal distribution of its pathological effects, in its multifarious lesions, and in its amenability to specific treatment; it is, moreover, contagious, but not infectious. Like the exanthematous fevers, syphilis has a definitive incubative period, a stage of efflorescence, and one of decline. Like them, also, one attack is almost always protective; but should the patient become reinfected, as occasionally happens, the disease either aborts or runs a very much less severe course.

The classification of syphilitic manifestations, in common use, is chiefly founded upon the order of their evolution. The disease passes through **primary**, **secondary**, **latent**, and **tertiary stages**, but the division between these is to some extent purely arbitrary, for the symptoms of each, especially in bad cases and in those imperfectly treated, may overlap. Thus symptoms which are commonly met with in the later stages of the evolution of the disease occur at an early period, or "precociously." In mild cases, when the patient is refractory to the poison, and especially those in which mercurial treatment has been begun early and has been pushed, the secondary stage and those subsequent to it may be completely suppressed, although such a happy event is rare, and the patient usually suffers from some, although perhaps slight, secondary lesions.

At the end of the period of incubation the primary lesion makes

its appearance, and lasts usually about six or eight weeks. Before this has passed away the secondary symptoms appear, and may manifest themselves slightly or severely, and remain intermittently or persistently present for one or two years, to be followed at a varying interval of time, or overlapped, by the tertiary phenomena. These may, however, never occur, or may do so many years after the patient has apparently been restored to complete health, the interval not having been marked by any symptoms referable to syphilis. In other cases the secondary and tertiary stages are separated by a varying interval of time, during which the patient may have had occasional symptoms or "reminders" (latent stage).

The primary stage usually lasts about ten or twelve weeks, and comprises the incubative period, the appearance of the initial lesion at the seat of inoculation, and the associated indolent enlargement and induration of the lymphatic glands in its neighbourhood.

The incubative period is usually from twenty-four to twenty-eight days; it may, however, be shorter or longer, but is very rarely prolonged beyond six weeks. During this period nothing abnormal may be noticed, and any abrasion through which inoculation has occurred may heal soundly without trouble; in other cases healing does not take place. If the patient has simultaneously contracted a soft sore, this may run its normal course, its base indurating, however, when the incubative stage of syphilis has passed.

At the end of the incubative period the first clinical manifestation of the disease occurs at the point of inoculation. This is the *initial lesion or Hunterian chancre;* it is always present, but from its situation may be overlooked or not diagnosed as a chancre; or its characters may be masked by the occurrence of phagedæna, the presence of soft sores, herpes, or some other source of irritation and inflammation; lastly, it may have healed before the patient comes for advice, and its position be only indicated by induration of the tissues and enlargement of the adjacent lymphatic glands.

The primary sore is usually single, but occasionally two or more are present. It is usually situated on the genital organs, but may be found anywhere, according to the manner of inoculation. Extra-genital sores may be difficult to diagnose, since they have often been treated under a misapprehension of their nature and their characters thereby altered. In doubtful cases, the surgeon should regard with suspicion any intractable, indurated sore with enlargement and induration of the neighbouring lymphatic glands, and should search carefully for secondary affections of the skin and

mucous membranes. In women a primary sore of the vagina or on the os uteri may readily escape detection.

A primary chancre does not always present the same features, but there are certain essential characteristics constantly present. Circumscription, dense induration, painlessness, and involvement of the lymphatics and glands are constant. In most cases the sore appears as an indurated ulcer or erosion, having a gristly cartilaginous feeling, and exuding a slight, sticky, highly contagious discharge which is never profuse or purulent unless the sore be irritated. Sometimes the whole glans penis or prepuce is the seat of general induration with patches of diffuse ulceration. A chancre occurring on the sheath of the penis or elsewhere on the skin, and if unirritated, is not accompanied by ulceration or loss of substance, but takes the form of a dense, hard, localised, and slightly raised papule, with a few dry scales of epithelium on the surface. Should such a sore be irritated, it will break down and form an indolent ulcer seated on a broad base of cartilaginous hardness. As a rule, in whatever form the primary chancre appears, it gives no trouble; but if irritated, acute inflammation with suppuration and perhaps phagedænic characters may ensue. The primary lesion may heal spontaneously, and rapidly does so under mercurial treatment; the seat of it, however, may break down again even after a long time. After healing there is, if ulceration has occurred, a small cicatrix, and the induration persists for some time.

About a week after the appearance of the initial sore, the lymphatic vessels leading from it are enlarged and cordy and the neighbouring glands become indurated. The glands are slightly enlarged, but quite painless; they remain discrete, since peri-adenitis is not excited. Suppuration never occurs unless some other poison, *e.g.* that of chancroid, is present.

The enlargement of the glands attains its maximum in two or three weeks and may persist for many months. The changes in the lymphatic system are due to an increase in the fibrous and cellular elements.

The secondary or exanthematous stage may last as long as two years, but rarely does so more than one. The lesions have a natural tendency to spontaneous cure, and rapidly disappear under mercurial treatment. The symptoms appear when the poison has induced general toxæmia, being disseminated throughout the body by the blood-stream, and has had sufficient time to induce pathological changes in the various tissues — that is, in about ten weeks from the date of infection, and six from the appearance

of the primary lesion; but this stage may be postponed for six months.

The secondary symptoms vary much in severity, and, as has already been mentioned, may in favourable cases be entirely, or almost entirely, suppressed. During this stage, and for some time after it, the patient is capable of conveying infection, but not of being himself reinfected. Preceding the appearance of the lesions characterising this stage in the evolution of the disease, there is often, especially in women and weakly subjects, general malaise, anorexia, and headache, with rheumatoid pains in the back and limbs. There may be some fever of a continuous or remittent type which, in rare cases, is very severe, and simulates typhoid. Fever is, however, more likely to occur with the appearance of the papular eruption. Anæmia from destruction of the red cells is constant, and may by itself prove a serious condition. Loss of fat and mental dejection are not uncommon. The manifestations of secondary syphilis chiefly consist of certain eruptions of the skin, analogous conditions of the mucous membranes, and affections of the hair, nails, eyes, and bones. The skin is especially affected by eruptions of the most varying clinical appearances (syphilodermata or syphilides), thus they may be macular, papular, vesicular, pustular, nodular, etc. Such eruptions have certain peculiar characters indicative of their syphilitic origin (see p. 177). The syphilides of the secondary stage are more superficial, more copious, more symmetrically arranged, and not so prone to ulcerate as are those which occur during the tertiary period of the disease. Mucous tubercles or condylomata occur at the muco-cutaneous surfaces, or in certain parts, *e.g.* the vulva, nates, and axillæ; these are merely the ordinary skin lesions modified by warmth and moisture consequent on their position (Fig. 35, p. 180). The appendages of the skin may also suffer from want of nutrition, the hair falls, and the nails may become brittle and furrowed, and the matrix may inflame. The mucous membranes, especially of the mouth and throat, are also affected by the eruption, which, in these situations, give rise to mucous tubercles, condylomata, or superficial grayish, often reniform, ulcers. General enlargement of the lymphatic glands is sometimes seen. The glands do not suppurate, neither do they indurate, as in the case of those associated with the primary lesion; they are slightly tender.

Iritis is by no means uncommon, and usually appears with the decline of the eruption; it is not infrequently bilateral, and in some cases there may be associated choroiditis, retinitis, or optic neuritis. Inflammation of the periosteum of an acute or sub-acute nature may

occur during the secondary stage; but it is always transient, and has no tendency to cause suppuration or death of the underlying bone, as is so frequently the case in the tertiary stage. The joints, bursæ, and synovial sheaths may also transiently inflame, sometimes with considerable effusion of fluid. Neuralgic pains, which are worse at night, are often experienced in the muscles, bones, and joints (syphilitic rheumatism). Syphilitic changes in the arterial system, especially affecting the intima of the vessels, are more likely to occur during this stage than at any other time throughout the disease. Affections of the nervous system (endarteritis, meningitis) may be met with, and it is noticeable that nervous lesions are especially liable to occur during the second year from the date of infection. The viscera are rarely affected, but they may be the seat of slight interstitial inflammation.

The latent period or period of relapses may be very prolonged even to as much as twenty or thirty years. During this period the patient may enjoy perfect health and may never show any evidences of the disease, but sometimes, especially if he should fall into ill-health, "relapses" or "reminders" make their appearance. Such symptoms usually take the form of superficial and often painful fissures or ulcers of the mucous membrane of the tongue or cheeks, or of skin eruptions similar to those which may be met with during the secondary stage, but being less profuse, more limited as regards the area of skin involved, and showing much less symmetry in their distribution.

The tertiary stage.—"By tertiary syphilis is meant those manifestations of the disease which are characterised (as distinct from the secondary symptoms) by the deposition of a syphilitic product, deep in the skin or mucous membranes, in internal organs, in the nervous system, in bones, blood-vessels, or muscles, and which, setting apart the different macroscopic forms in which they appear, agree in not tending to resolution, but in leaving, through sloughing or ulceration, or organisation into fibrous tissue, permanent changes which, in the form of scars or contraction of the tissue, lead to consequences more or less serious according to their situation" (Haslund).

The real nature of tertiaries is disputed; Hutchinson regards them as sequelæ, comparable to such as may occur after any other exanthematous fever, *e.g.* scarlet fever, and others think they are the outcome of a specific cachexia produced by the saturation of the system with the syphilitic toxine. That tertiaries are not dependent upon the action of the syphilitic germs which have

remained dormant for a long period until circumstances favour their activity, seems to be indicated by the fact that during this stage the patient is incapable of conveying infection, unless we are prepared to suppose that such germs have undergone some change whereby they are inimical to the host himself, but are incapable of producing syphilis in another.

Whereas in the secondary stage the patient can convey the contagion to others and to his offspring, but is immune to fresh infection, in the tertiary he cannot transmit the disease, but may acquire a fresh dose of poison (reinfection); moreover, the secondary lesions tend to spontaneous cure, while tertiary symptoms tend rather to persist and spread, and do not show that symmetrical arrangement which is so characteristic of the secondary stage.

Tertiary symptoms may occur any time after two or three years from the date of infection; the essential feature of this stage is the formation of inflammatory masses known as gummata, with which is associated diffuse interstitial chronic inflammation; in most cases these two processes are combined, the gummata resulting from the greater intensity of the process in certain parts.

Gummata are tumours of syphilitic origin due to localised inflammation. A gumma is composed of small, round, densely packed cells, poorly nourished by vessels of new formation, which themselves soon undergo syphilitic changes, rendering them inadequate to supply sufficient nourishment. As the gumma increases in size, it displaces and causes atrophy of the tissues; but since growth is in excess of destruction, a definite tumour or syphiloma is formed, rarely exceeding the size of a walnut unless adjacent gummata have fused. In consequence of the poorness of the vascular supply, gummata are very prone to degenerate and soften, and unless the process be arrested and absorption favoured by treatment, the overlying skin or mucous membrane (in the case of gummata so situated) becomes involved, and on its giving way, the granular débris mixed with pus is discharged and a characteristic sore is produced. Visceral gummata very rarely soften and break down; they may undergo more or less complete absorption, their situation being marked by a small puckered cicatrix. Calcification is rare.

To the naked eye a gumma superficially resembles a patch of caseous tubercle, but the pale yellow, degenerated area is more dense and elastic unless softening has occurred. The mass is circumscribed and surrounded by a more or less dense, grayish zone of chronic inflammatory tissue, which forms an imperfect kind of capsule. In tubercular masses, nodules of tubercle are seen

surrounding the central mass, and to the breaking down of these the increase in size is due. Gummata are vascularised; tubercular patches are not.

Gummata progress slowly and painlessly, and form rounded elastic tumours, which soften and involve the skin as degeneration advances. In the early stages they may be mistaken for definite new growths, especially lipomata; but their situation and want of lobulation, together with other evidences of syphilis and the effects of treatment, will usually be sufficient to avoid error. When softening has occurred, a gumma may be mistaken for a subcutaneous, tubercular abscess. Broken-down gummata form steeply-cut, circular, or crescentic ulcers, with steep and often undermined edges; the base is covered with a tough adherent slough like wet chamois leather. When the slough separates, an ulcer secreting thick tenacious discharge and showing no tendency to improve under ordinary local measures results. Healing by granulation is very rapid under iodide treatment, a soft, supple, white, non-contracting scar being left.

The tertiary inflammation may give birth to new scar tissue, or may ultimately break down, soften, and ulcerate. Tertiary lesions have a strong tendency to recur again and again in the same tissue; thus, if the skin be affected, a relapse of the symptoms will probably affect some part of it. The skin is far more commonly affected by tertiary lesions than is any other tissue, and next in order of frequency the bones, nervous system, mucous membranes, and internal organs. It is in the nervous system and the internal organs that the tertiary phenomena are most disastrous in their effects.

The skin and mucous membranes may be the seat of serpiginous ulcers which tend to recur again and again; similar inflammatory and destructive changes may occur in the bones leading to chronic periostitis and osteitis, caries or necrosis, and in the viscera to sclerotic changes which, in the case of the nervous system, may induce paralysis or even insanity. The joints, synovial bursæ, and tendon sheaths may all suffer. As regards the intestinal tract the rectum is most commonly affected, and stricture usually results as the new scar tissue contracts. Tertiary syphilis may also affect the muscles, but rarely does so, except in the case of the tongue.

SYPHILITIC AFFECTIONS OF THE SKIN

Syphilitic eruptions, known as syphilides or syphilodermata, have their non-syphilitic prototypes, but their specific origin confers

upon them certain peculiarities of diagnostic import. Such peculiarities vary with the situation of the eruption, and according to whether it be kept dry and unirritated, or moist and warm.

Special features of the syphilides.—Syphilitic eruptions present the following diagnostic features:—

(1) They are, to a large extent, composed of dense collections of cells, and are consequently solid and firm to the touch, and do not disappear on pressure; the macular syphilide is, however, exceptional in these respects (p. 178).

(2) Their course is indolent, and they tend to recur, perhaps again and again, for months—sometimes for years.

(3) They tend to disappear spontaneously after a time, and rapidly do so under the influence of mercury in the early stages of the disease and of the iodides in the tertiary.

(4) The individual spots have a rounded outline, and the earlier maculo-papular or papulo-nodular lesions are irregularly distributed. They undergo centrifugal extension, so that they tend to become ringed, and by confluence may produce varied figured patterns, or become crescentic. The later lesions tend to be grouped by the formation of fresh lesions around the sites of the older disappearing ones, and in this way the eruption may creep, in the form of segments of circles, across a region (serpiginous spread).

(5) The eruptions are copious, widely distributed, symmetrical, and superficial during the early secondary stage, but with remoteness of the date of infection they become less copious, more localised, asymmetrical, and tend to affect the deeper layers of the skin, the subcutaneous structures, and the viscera.

(6) The earlier lesions are frequently multiform or polymorphic in character. This polymorphism may be due to the simultaneous presence of various types of eruption, *e.g.* the macular, papules of varying size, vesicles, pustules, etc., or to the various stages which such eruptions undergo, or to their modification in special regions, *e.g.* a papular eruption tends to become crusted about the forehead and scalp, to form condylomata about the anus, vulva, or other warm and moist regions, and to form mucous patches in the mouth.

(7) The lesions are of a coppery-red or raw-ham colour from the escape of the red blood cells, and when they die away, leave marked staining.

(8) The eruptions do not as a rule cause pain, itching, irritation, or any subjective sensation.

(9) The different syphilides show a special predilection for certain sites, thus the macular form especially affects the abdomen, the early papular eruption selects the flexor rather than the extensor aspects of the limbs, and the mucous membranes of the mouth, throat, and larynx are very prone to be affected.

Varieties.—Syphilitic eruptions may be macular, papular, vesicular, pustular, bullous, pigmentary, and nodular or gummatous, but they are all due to a process of inflammation of greater or less intensity, involving the superficial papillary layer of the skin, or extending more deeply, and hence they tend to merge the one into the other. There is round-celled infiltration in the immediate neighbourhood of the blood-vessels, the round cells being escaped leucocytes with which are mixed a few red cells to which the characteristic pigmentation is due. If the vascular hyperæmia is circumscribed, and the cellular infiltration is slight, the macular syphilide results; when the infiltration of the papillary body is marked, papules of varying size occur, and such may, from the intensity of the process, become vesicular, bullous, or pustular. In the most marked cases of infiltration in which the process extends deeply, perhaps involving the subcutaneous connective tissue, the nodular or gummatous syphilide results, and by the disintegration of the inflammatory tissue ulceration follows, and may assume serpiginous characters owing to central healing with fresh invasion at the periphery. When absorption occurs, it is often many months before all traces of the eruption disappear, the pigmentation being very often long persistent.

The macular or roseolar syphilide.—This, the earliest and most constant of the eruptions, usually makes its appearance about six weeks after that of the initial lesion, but it not infrequently escapes notice. The eruption may be profuse or scanty, and chiefly affects the anterior aspect of the trunk, especially the abdomen; it gradually attains a maximum in about a fortnight, and within another week begins to fade, but relapses are common. The maculæ vary in size, being usually that of a split pea, but sometimes as large as a shilling. They are at first of a pale rose-red colour and fade on pressure, but later on the colour is more livid, and as the eruption fades, is replaced by coppery discoloration. There may be, but rarely is, slight desquamation. The macular and papular syphilides are usually concurrent, and with the appearance of the former, there is frequently erythema of the fauces, and perhaps of the laryngeal mucous membrane.

The papular and papulo-squamous syphilide.—The

papules are due to cellular infiltration of the papillary body, and present themselves under a great variety of forms. The individual lesions are solid, strictly circumscribed, raised above the surface, and of a raw-ham or coppery colour; the epithelium usually proliferates and separates from the papule, its remains showing as a silvery collarette. The papules may be large or small, discrete or fused, and are often arranged in circular or crescentic groups. It is important to note that the character of the eruption is modified by its situation; thus in the palm and sole the proliferation of the epithelium is a marked feature, and the papules become hard and horny on the surface (papulo-squamous syphilide), and are not infrequently painfully fissured; papules in warm and moist situations, *e.g.* round the vulva or in the mouth and throat, form condylomata or mucous tubercles which will be referred to later on.

The small or miliary papule occurs in corymbose clusters seated round the hair or sebaceous follicles, and varies in size up to that of a lentil; the papules are distinctly shotty, and are at first pinkish in colour, but soon assume the characteristic coppery tint and leave slight staining as they die away. The eruption may appear in successive crops, chiefly affecting the trunk and limbs; it may last for six weeks or as many months. Sometimes the miliary papule suppurates, and thus passes into the acneiform pustular eruption (see p. 181).

The large papular or lenticular syphilide is very common, and occurs coincidently with, or very soon after, the macular eruption; it is, however, very liable to relapse, and consequently may be present years after infection. The papules, which may be as large as a sixpence, are rounded and flattened; they make their appearance in successive crops which gradually fade away, although evidence of their occurrence often persists in the form of coppery staining for many months. This form of eruption may be universally distributed, but shows a special predilection for the nape of the neck, the forehead along the line of the hair (corona veneris), the mucous membrane of the mouth, the flexor aspects of the limbs, and for the palms and soles; the last two situations are often favoured by late relapses of the eruption. The epithelium frequently desquamates and covers the surface of the papule with silvery scales (squamous or scaly syphilide), so-called syphilitic psoriasis.

Moist papules, mucous patches, or **condylomata** (Fig. 35, p. 180) result when the papular eruption affects warm and moist regions. They are consequently especially met with round the anus, between

the folds of the buttocks, round the vulva, in the axillæ, in the sulcus beneath the breasts, round the mouth, especially at the angles, and between the toes. The papules are broad, flattened, soft, and often confluent; the sodden surface epithelium desquamates, and the mucous plaques secrete a thick, tenacious, offensive, and highly contagious secretion. This secretion is auto-inoculable, and hence mucous tubercles give birth to others on parts with which they may be brought in contact, *e.g.* the inner side of the thigh and labium, and the folds of the nates. If the individual plaques fuse, a considerable area may be affected, and owing to irritative overgrowth of the papillæ the mass increases in thickness, and has a

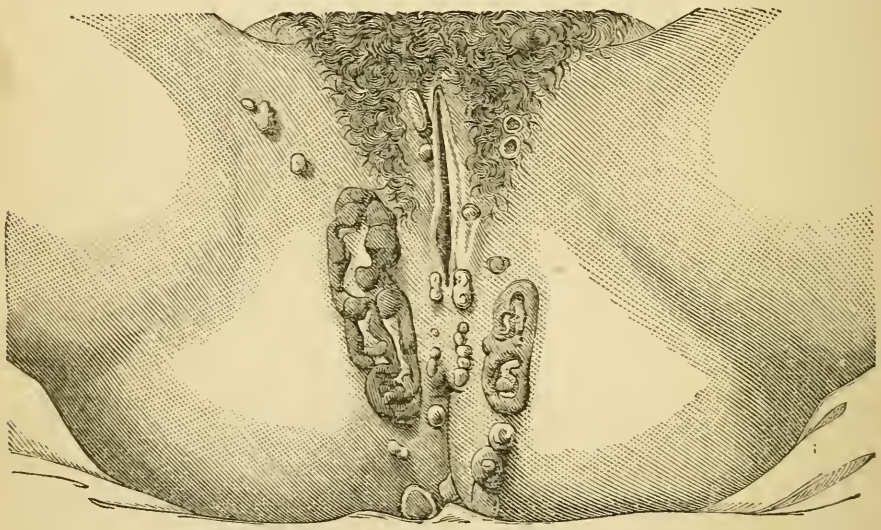

FIG. 35.—Syphilitic ulcers and condylomata (Jullien).

fungous appearance. Sometimes, *e.g.* about the angles of the mouth, mucous patches become fissured, or they may ulcerate, and especially tend to do so when they are situated between the toes (rhagades). The resulting ulcers are very foul, but rapidly heal under mercurial treatment. Mucous tubercles in the mouth and throat lead to characteristic ulceration, chiefly affecting the tonsils, palate, fauces, sides of the tongue, and buccal, mucous membrane (see p. 185). When it occurs on the palms and soles the papular eruption is modified by the thickness of the epidermis, and appears as the so-called palmar psoriasis. The hyperæmic areas are covered with proliferated and desquamating epithelium, which, separating at the margins, tends to curl up, but remains adherent at the centre of the patch; painful fissures and

cracks are common, and especially occur along the normal lines of flexure of the skin. Palmar psoriasis tends to spread peripherally, but may heal in the centre; it is often asymmetrical, is very persistent and liable to relapse, and may be met with years after infection. Its presence is of great diagnostic value. In some cases the epithelium does not desquamate, but, continuing to proliferate, forms a dense, hard, and horny patch.

The vesicular syphilide.—This form is extremely rare, and usually occurs as the eczematous variety, but varicelliform and herpetiform syphilides are described. The eczematous syphilide occurs in successive patches or groups of very small vesicles situated on coppery areas, which are formed by the coalescence of the coppery rings which surround each individual vesicle. The vesicles contain clear, but sometimes cloudy, fluid which dries up and leaves an adherent scale which, on separating, leaves coppery staining. The vesicular eruption is usually an early manifestation, and may become pustular.

The pustular syphilide.—The development of pustules usually occurs as a late manifestation of the disease, and is very rarely met with unless the patient's health be markedly deteriorated. The pustular stands in an intermediate position between the vesicular and bullous eruptions. The pustules vary considerably in size, but when they occur early they are usually small; they may be localised or diffuse, and are sometimes grouped; they vary in shape and in their distribution, and each is surrounded by a coppery ring. When the pustules burst they become covered with a crust, beneath which ulceration extends. The various forms of pustular eruption are named according to the non-syphilitic eruption which they mimic. Thus they may be acneiform, varicelliform, varioliform, ecthymatous, etc.

The acneiform syphilide, as already stated, is a suppurating form of the small papular eruption, with which it is often associated (see p. 179). This eruption usually occurs early, and is not infrequently universally distributed, although showing a preference for the face and shoulders. The pustules are small, and are seated on coppery papules; as the pus dries, a thin scale is left which falls and leaves a small scar.

The impetiginous and ecthymatous syphilides occur as large pustules seated on coppery papules. The lesions soon become covered with crusts, beneath which ulceration may extend widely. The impetiginous form is usually localised to the face, scalp, and genitals, but respects the limbs, whereas the ecthymatous, although

it may occur anywhere, shows a predilection for the shoulders, back, and limbs. The ecthymatous syphilide may occur early or late, the early lesions being usually smaller and more superficial than are the late, which occur coincidently with the tertiary lesions of the bones and viscera; the late form is marked, moreover, by the great tendency to the formation of adherent crusts on the surface of ulceration (**pustulo-crustaceous syphilide—rupia**, Fig. 36). Rupia is diagnostic of acquired syphilis; it never occurs in the inherited disease, and only attacks patients in vitiated health, which may be further impaired with the advent of the eruption. As the ulceration extends in area, so each succeeding lamina of crust is larger than the last, and hence the scab assumes the characteristic, conical, or limpet-shell shape; if this be removed, a deep ulcer, surrounded by a red areola, is disclosed, which will, unless constitutional treatment be adopted, gradually spread, and may assume serpiginous characters. When the ulcer heals a deeply stained scar results, but in the course of time this becomes white, thin, shiny, and supple, and it may for some time be surrounded by a pigmented ring.

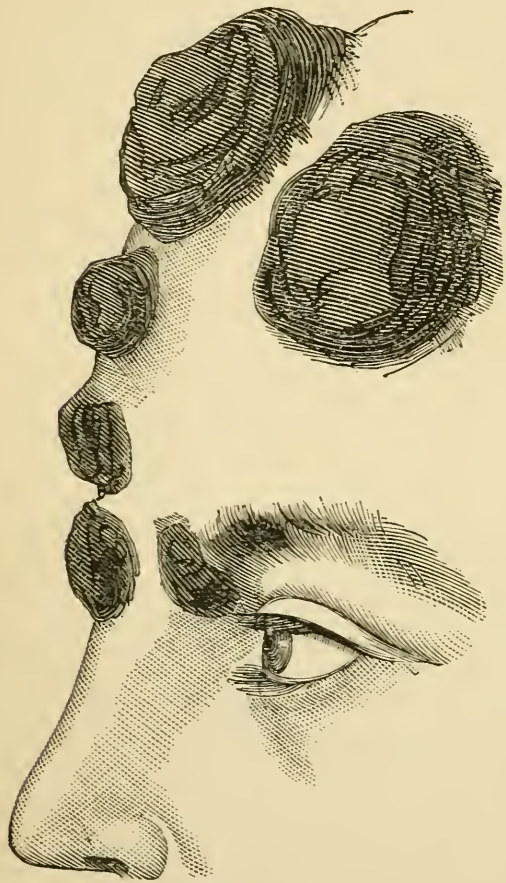

FIG. 36.—Rupia syphilitica (Jullien).

All the pustular syphilides are liable to persist for a long time and to relapse; they must in all cases be regarded as serious manifestations, indicating as they do, either a naturally weak constitution, or one which has been undermined by privation, debauchery, or some other cause; they are also likely to occur in malignant forms of syphilis in which the patient has proved very refractory to mercurial treatment.

The bullous syphilide is practically identical with the large pustular form, and gives rise to eruptions like ecthyma or rupia. Syphilitic pemphigus (Fig. 37) has occasionally been noted as occurring in the acquired disease, but it is essentially a manifestation of the hereditary. The bullæ form on the palms and soles.

The pigmentary, leucodermia-like or dappled syphilide.—This purely pigmentary change is a secondary manifestation, and occurs almost exclusively in women, affecting the sides of the neck, although it is occasionally seen elsewhere. The eruption consists of discrete, rounded, *café-au-lait* coloured stains, which may be very

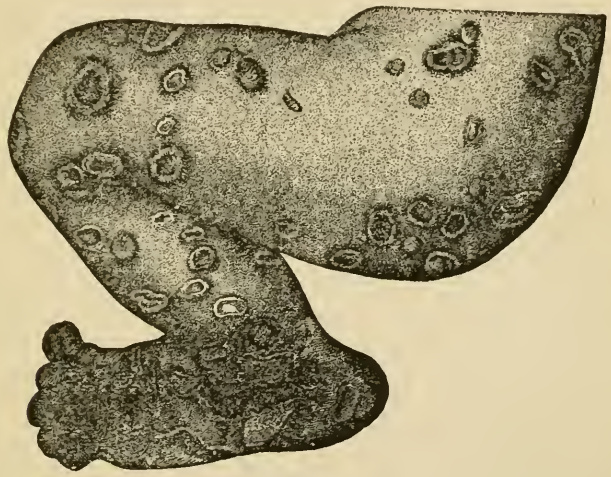

FIG. 37.—Pemphigus and circular ulcerations in an infant with congenital syphilis (Follin).

faint, so that they can only be seen in certain lights; they are not raised above the surface, and do not desquamate. The spots may attain the size of a sixpence, and by confluence may give rise to a peculiar lace-work pattern enclosing areas of unaltered skin which looks by contrast whiter than normal. The affection is very persistent, and resists anti-specific treatment; it is not quite certain that the eruption should be regarded as peculiar to syphilis, and that it may not occur in persons of depraved health from other causes. The pigmentary syphilide is not to be confounded with the staining which remains when other eruptions have died away; it is also to be distinguished from tinea versicolor, in which disease the pigment is *on* and not *in* the skin.

The nodular or gummatous syphilide.—This lesion belongs essentially to the tertiary stage of the disease, but may occur

at the end of the second year; it is especially met with in cachetic patients, whose health is seriously broken down. The nodules may be as large as a pea, bean, or walnut, and may implicate the deeper parts and the subcutaneous tissue (subcutaneous gummata). They are rounded or oval in shape, solid and dense to the touch, and coppery or brownish in colour. If the eruption occurs early in the evolution of the disease it is liable to be widespread and symmetrically distributed, but during the later years it is more scattered, less likely to be symmetrical, and by confluence large patches may be formed. The nodules have a special tendency to appear in clusters, and as the old ones die away others appear at some part of the periphery, and so circles, or segments of circles, sometimes reniform, may be formed. In this manner the eruption will wander across a particular region, as in some cases of lupus (*serpiginous spread*). Relapses are common, successive crops of nodules appearing from time to time, and although no part is exempt, yet they especially favour the face, forehead, neck, and arms. When the nodular syphilide has reached a maximum, it may die away or ulcerate, and thus two forms, the ulcerating and the non-ulcerating, are usually described. In the non-ulcerating form the nodules gradually undergo absorption, and the subcutaneous tissue may participate, so that a scar is left which gradually increases in extent if the eruption, by spreading at its periphery, assumes serpiginous characters. Sometimes the nodules project considerably beyond the surface, and may be crusted or covered with desquamating epithelium. The nodular syphilide is especially prone to ulcerate if the health of the patient is vitiated; the ulceration sometimes extends widely and deeply, and with considerable rapidity; at others it progresses more slowly and tends to spread in superficial area rather than in depth, and to assume serpiginous characters. The superficial ulcers are covered with a dirty brown crust; they assume an oval or crescentic shape, healing in the centre and spreading at the periphery, so that the lesion may travel a long distance from the point of its initial manifestation. When healing occurs the cicatrix is livid, and may remain so for a very long time. When the process of ulceration extends deeply a characteristic gummatous ulcer results, and in certain parts, *e.g.* the nose, considerable destruction may ensue. These gummata are rarely numerous, and are especially met with about the face, nose, scalp, and in the neighbourhood of the knee and elbow, but they may occur anywhere.

SYPHILITIC AFFECTIONS OF THE MUCOUS MEMBRANES

With the appearance of the macular syphilide there is general erythema of the fauces, palate, and tonsils, and sometimes of the laryngeal mucous membrane, accompanied by some slight discomfort and dryness of the throat with hoarseness, but no actual pain. During the period of evolution of the papular eruption this involves the mucous membrane of the fauces, tonsils, sides of the tongue, floor of the mouth, and buccal aspect of the lips and cheeks, but the lesions become altered in appearance. On the mucous surfaces the lesions appear as small, reniform, or crescentic superficial ulcers, which have a dirty ashy-gray base, and a dead white margin of sodden epithelium; these ulcers are usually symmetrical, and do not show any tendency to spread either in superficial area or depth, nor are they usually very painful, although they cause some stiffness and soreness of the throat. The patient may sometimes be unconscious of their presence; the secretion from these ulcers renders the saliva highly contagious. Sometimes there is considerable overgrowth, and the patches project more or less beyond the surface (mucous tubercles, condylomata); this is very likely to happen at the sides of the tongue and angles of the mouth. Similar patches are also coincidently present at the vulva and round the anus (Fig. 35, p. 180); but whether they occur in any other part of the intestinal tract is unknown.

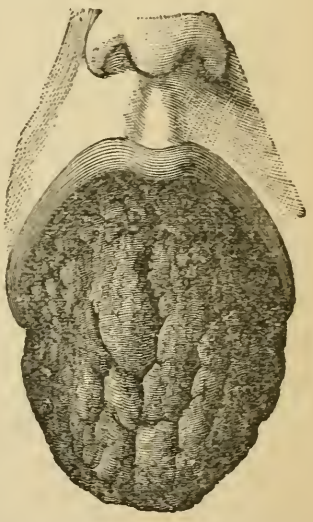

FIG. 38. — Extensive fissuring of the tongue following syphilitic ulceration (Follin, after Clarke).

Tertiary ulceration is commonly met with about the throat, tongue (Fig. 38), lips, cheeks, and larynx, and (especially in women) the lower end of the rectum and the anus may be affected.

SYPHILITIC AFFECTIONS OF THE HAIR—SYPHILITIC ALOPECIA

The hair may be affected in consequence of general want of nutrition and cachexia, or as the result of some syphilide of the scalp. The hair loses its lustre, becomes brittle, dry, and falls. The loss of hair may be patchy and scanty, or may be general and profuse; it generally occurs during the first year of the disease, but

sometimes at later periods; under treatment, and especially with general improvement in the patient's health, the hair quickly grows again, but if its loss is due to ulceration of the scalp, baldness will be permanent.

SYPHILITIC AFFECTIONS OF THE NAILS—ONYCHIA—PERIONYCHIA

These affections are due to want of nutrition, or to inflammatory or ulcerative lesions affecting the matrix. In the case of onychia the nails (many of which may suffer) become longitudinally furrowed, brittle, jagged, and are eventually cast; sometimes there is a deep groove across the nail at the base of the semilunar furrow. Perionychia or inflammation of the matrix of the nail is associated with the appearance of some form of syphilide; the nail is deprived of its nutrition and may be shed, but usually grows again without marked deformity. Sometimes perionychia is associated with considerable ulceration, discharge, and pain. These lesions usually occur during the first year, but like those affecting the hair, may do so at a later period.

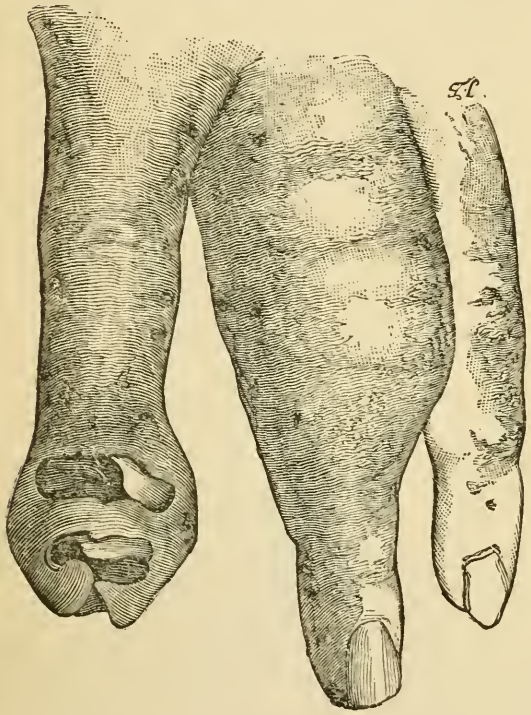

FIG. 39.—Syphilitic dactylitis and onychia. (Follin, after Blum.)

SYPHILITIC AFFECTIONS OF THE LYMPHATIC GLANDS

About a week or ten days after the appearance of the primary chancre, the lymphatic glands in the neighbourhood undergo indolent enlargement, unaccompanied by pain or tenderness, or by any signs of acute inflammation; they do not suppurate, and after remaining for a time *in statu quo*, gradually resume their normal

size in from four to six months. It not infrequently happens that at the period of greatest intensity of the early syphilide, there is a general enlargement and tenderness of the lymphatic glands throughout the body, but unlike that occurring in association with the primary sore this enlargement is transitory in nature, and is unaccompanied by induration. When the throat is affected there is often subacute sympathetic inflammation of the submaxillary lymphatic glands, and occasionally of those beneath the sterno-mastoid. It is to be noted that the tertiary eruptions and ulcerations are unaccompanied by glandular involvement.

SYPHILITIC AFFECTIONS OF THE MUSCLES AND BURSÆ

Rheumatoid pains are not uncommon in the early secondary stage, but gummatous lesions are rare, the muscular substance of the tongue being their most common site (see Diseases of the Tongue, vol. iii.), in which situation care must be taken to diagnose the affection from cancer.

The bursæ, especially that in front of the patella, may be the seat of gummatous inflammation, which may originate in the connective tissue surrounding it.

SYPHILITIC AFFECTIONS OF THE JOINTS

In the early secondary stage the patient occasionally complains of rheumatoid pains in the joints. In rare instances one of the larger joints, usually the knee, is the seat of transient acute synovitis and is filled with fluid; there is, however, as in the case of the earlier periostitis, no tendency to suppuration, and the condition quickly subsides under mercurial treatment, and leaves no traces behind it. During the tertiary period **peri-synovial gummata** may affect the fibrous tissues round the knee, and less frequently round the elbow joints; the gummata may burst on the surface, but rarely do so into the joint, although there is not uncommonly some associated synovitis. This manifestation is much more likely to occur in women than in men, runs a chronic course, and is very liable to relapse even after apparent complete cure.

Chronic gummatous synovitis may occur during the tertiary period, but is very rare. It chiefly affects the knee, and very closely simulates white swelling (the syphilitic pseudo-white swelling of Richet). The condition runs a very chronic course and causes the patient but very little inconvenience. It is very amenable to

anti-syphilitic remedies, but if these be not administered, destructive arthritis may ensue, or a certain amount of fibrous ankylosis may result.

Lastly, the joints may be secondarily involved by articular caries due to gummatous infiltration of the cancellous tissue of the bones entering into their formation (see also chap. vi. vol. iii.).

SYPHILITIC AFFECTIONS OF THE BONES

With the onset of the exanthematous stage, pains in the bones of greater or less severity are not uncommonly experienced in association with neuralgia of the muscles and joints (syphilitic rheumatism). Periostitis, slight and transient in nature, and having no tendency to suppurate, or very little to lead to the formation of new bone, is also common. In the late secondary and tertiary stages, chronic periostitis with the formation of nodes, sclerosing osteitis, and necrosis are common manifestations of the disease. Although any bone may be affected, those superficially placed are specially prone to suffer; thus the calvaria, palate, bones of the nasal fossæ and the tibiæ are frequently affected, and in the first situation the consequences may be grave, owing to involvement of the inner table and consecutive disease of the meninges and brain. Syphilitic dactylitis closely resembles the tubercular variety (see Fig. 39, p. 186).

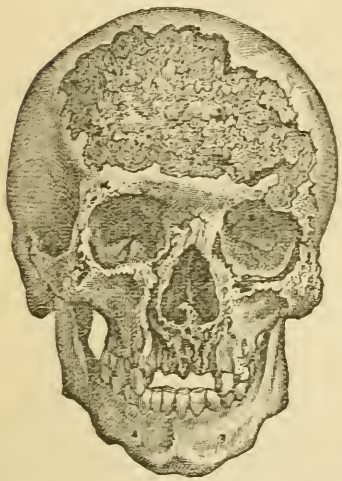

FIG. 40.—Syphilitic caries and necrosis of the bones of the skull. (Musée Dupuytren. Follin.)

Syphilitic inflammation of the periosteum or bones may run a very chronic course, being chiefly characterised by the formation of new osseous tissue, but sometimes the course is more acute, and the gummatous tissue softens, breaks down, and abscess results. The special affections of the bone met with in hereditary syphilis will be subsequently referred to (see p. 203).

SYPHILITIC AFFECTIONS OF THE BLOOD-VESSELS

The intima of the arteries is especially affected. When arterial disease occurs, it is in most cases early in the secondary stage, but

such changes may, like the other lesions of this period, quite clear up. During the late secondary or tertiary stage, atheroma of the larger vessels and endarteritis of the smaller (especially those of the brain) may occur. Syphilitic endarteritis is characterised by proliferation of the intima at one side of the artery, which consequently bulges into the lumen and narrows it. Gradual infiltration of the coats renders them thick and rigid. As a result of such changes, aneurism, thrombosis, or gradual obliteration of the vessels may occur, and in the case of the brain and cord serious nervous lesions, such as hemiplegia and paraplegia, may result from the consequent interference with the nutrition of the nervous elements.

SYPHILITIC AFFECTIONS OF THE NERVOUS SYSTEM

During the last quarter of a century many observers have shown that the first two years after infection exhibit an extraordinary predominance, as far as the development of nervous symptoms is concerned, over any similar period during the whole course of the disease. They prove that nearly one-half of all the nervous affections met with in syphilis appear during the first two years, Such lesions may, however, appear very many years after infection, and so great is the connection between antecedent syphilis and nervous diseases that, in all cases of the latter, a history of the former should be diligently sought for. Nervous lesions may be occasioned by disease of the basal arteries, by sclerosis or gummatous deposits in the meninges, by gummatous deposits, thickening or sclerosis of individual nerves (notably the third or oculo-motor), which lead to local paralysis, and lastly, by the occurrence of meningo-myelitis or gummata in the nervous tissue, which probably originate in the peri-arterial structures. Tabes dorsalis and general paralysis of the insane may be specially mentioned in connection with this subject.

SYPHILITIC AFFECTIONS OF THE EYES

Iritis is not uncommon during the secondary stage of syphilis, and occurs coincidently with the skin lesions. Both eyes are frequently involved. The inflammation is chiefly characterised by a marked tendency to plastic infiltration; small beads of lymph are seen on the posterior surface of the cornea, and similar nodules may be present on the iris. The usual symptoms of iritis are

present, but the pain and photophobia are notably less than in non-specific cases. Interstitial keratitis has been occasionally met with in the acquired disease. In the late secondary, latent, or tertiary periods inflammation may occur in the deeper structures. Disseminated or diffuse choroiditis, associated with retinitis, is the most common; the disease is very insidious and persistent. Papillitis and interstitial retro-bulbar optic neuritis may also be met with.

Ocular paralysis from involvement of the third nerve or its nuclei, or in association with meningitis, is not rare in the late secondary or tertiary stages; the muscles affected vary, the pupil may be dilated, and the levator palpebræ superioris paralysed; drooping of the upper lid is very suggestive of syphilis.

SYPHILITIC AFFECTIONS OF THE VISCERA

Lesions of the viscera are usually late manifestations. The lesion consists in diffuse or localised interstitial inflammation. In the localised form new growths (gummata or syphilomata) make their appearance as they do in other tissue, but when affecting the internal viscera they do not show the same tendency to soften and break down, but rather to undergo fibrotic changes. Of the viscera, the liver and body of the testis are most frequently affected; but in the latter situation, softening with gradual involvement of the superficial structures and subsequent formation of a gummatous ulcer are common. When the abdominal viscera are the seat of syphilitic inflammation, the fibrous capsule and investing peritôneum are thickened from the same cause. The lungs are sometimes indurated as in chronic phthisis, and cavities may result from breaking down of the gummata; the change is usually limited to one lobe. The larynx is often the seat of gummatous inflammation, which may lead to necrosis of the laryngeal cartilages and considerable destruction, with subsequent contraction and obstruction. Syphilitic stricture of the rectum is almost confined to women.

PROGNOSIS AND TREATMENT OF ACQUIRED SYPHILIS

Prognosis.—In modern times syphilis is by no means the formidable disease which it was when first introduced into Europe, but even now, if it be introduced into virgin communities, it runs a very much more severe course than under ordinary circumstances is the case. The modern alleviation in the course of the disease

is no doubt in large measure due to great advancement in diagnosis and greater skill in treatment; but it would seem probable that there is, as it were, some degree of immunity conferred on those born in communities in which syphilis has been long endemic. Syphilis is certainly a curable disease, and the fact that re-infection occasionally occurs is the strongest argument in support of this statement. In the course of time the disease tends to expend its energy and to die out, and this even without mercurial treatment. We should, therefore, naturally expect that a prolonged and judicious course of this drug would hasten recovery and eliminate the poison from the system. Berkeley Hill says: "Although we cannot assure a patient that he is cured beyond possibility of a relapse, experience shows that if a sufficient and properly prolonged course of treatment has been carried out from an early period, the patient may expect to go through life with scarcely more appreciable risk than one who has never had the disease." As already stated, in discussing etiology, some persons prove to a certain extent refractory to the poison of syphilis, whereas others are especially prone; but it is noticeable that in all cases of syphilis, as indeed of any other general disease, the severity of its course is in great measure dependent upon the general health and no less upon the habits of the victim. In the majority of cases no serious symptoms occur, and the patient remains practically free from all manifestations of the disease after the first six months provided he has been properly treated; in some cases the manifest lesions disappear even earlier than this, and of this fact the patient should be made acquainted, otherwise he may neglect to continue under treatment. Chronic alcoholism, renal disease, tubercle, and a strumous diathesis add to the gravity of the prognosis, since such conditions materially impair the general health, and the two first interfere with assimilation and the destruction of waste products; there is probably no condition which is more serious in a case of syphilis than chronic alcoholism, in the rich it is too often associated with a generally irregular habit of life, and in the poor it is indulged in at the expense of proper food, lodging, and clothing. In patients over the age of forty, when the disease is contracted, it is liable to run a long and intractable course. But little real information as regards the ultimate prognosis can be gained from the appearance of the primary lesion and the secondary manifestations, although some consider that a short incubative stage, followed by a chancre which is extensively indurated and prone to ulcerate, heralds a severe form of the disease. Indeed, in many cases a small insignificant sore is

followed by severe and persistent symptoms; this may, of course, be possibly due to the fact that such a sore being lightly regarded by the patient is neglected. Extra-genital chancres may also be followed by severe symptoms, but this again is dependent upon the fact that they are often difficult of recognition, and hence mercurial treatment is delayed until an unmistakable syphilide clears up the diagnosis; there is no reason to suppose that a chancre, say on the finger, should be more virulent than one on the penis, provided both are placed under the same conditions as regards treatment. With regard to the ultimate prognosis from the appearance and persistence of the secondary lesions it may be generally stated that, if these be marked, tertiary lesions will either be very slight or absent, perhaps because the virulence of the poison is in large measure expended on these lesions, but no doubt also because it is just in these cases that treatment is most energetically and assiduously carried out for a lengthened period. General glandular enlargement is often indicative of a severe attack. Tertiary symptoms do not occur in more than about 5 per cent of all cases among better class patients under private treatment, but in the case of the poorer hospital out-patients about 15 per cent suffer.

Treatment.—The **preventive treatment** consists in personal cleanliness and avoidance of contagion. Circumcision doubtless diminishes the risk of infection of all venereal diseases, and it may here be properly pointed out that this slight operation is one which might be very wisely performed in many children in whom it is neglected. There is little doubt that the Jews instituted circumcision on hygienic rather than on religious grounds. The reinforcement of the Contagious Diseases Act is much to be desired.

The *contraction of marriage*, even under the most favourable circumstances, should never be permitted until the expiration of two years from the date of inoculation, and even then no person should be allowed to marry if active signs of secondary syphilis are or have been present within the past six months, no matter how long after infection they may appear, for transmission to the offspring has been known after many years. It is probable that the power of transmission of the disease to their offspring lasts longer in women than in men.

Curative treatment.—The curative treatment of syphilis to be entirely satisfactory must be undertaken from two standpoints: (1) The poison itself must be destroyed by the use of mercury, and its later effects removed by the iodides; and (2) the patient must receive

such treatment as is calculated to counteract or minimise any condition of ill-health or any peculiar diathesis of which he may be the subject, and which, if left untreated, may render him, so to speak, a more fitting culture medium for the syphilitic virus. It must further be remembered that the knowledge that he is syphilitic, and the exaggerated dread of the ultimate consequences of the disease, which he probably entertains, added to the depressing effects of a prolonged mercurial course, may further accentuate any condition of ill-health of which he may be the subject.

During the primary and secondary stages of syphilis, mercury is a specific, and tertiary lesions are equally influenced by iodides. The non-mercurial treatment of syphilis is practically no treatment at all, and the improvement which will undoubtedly occur, and which is attributed, by its supporters, to this method is merely due to the curative effects of time, for, as has already been stated, syphilitic lesions tend to undergo spontaneous cure, and such improvement would have equally ensued had no treatment been adopted. The iodides of potassium, sodium, and ammonium are especially useful in the tertiary stage, but in some cases may be used with advantage during the secondary. The vegetable bitters, cod-liver oil, maltine, iron, quinine, Fellows' syrup, or the mineral acids may be given with advantage to women, and to strumous or weakly patients, and should usually be administered to all when the mercurial course is temporarily intermitted; but these drugs are adjuvants only, and are in no sense to be regarded as specifics.

Under ordinary circumstances no special alteration in the patient's habits and mode of life is necessary, but if these are of an unhealthy nature, a more healthy regime must be prescribed; while the rich and luxurious must be warned against over-indulgence, the dietary of the poor must be made as good as circumstances permit, and alcoholic excess must be strenuously interdicted in both. As a rule, malt liquor is not well borne by those undergoing a mercurial course, but if the patient is feeble and his health poor, a glass of stout in the middle of the day may be beneficial. Fruit and green vegetables sometimes occasion gastro-intestinal disturbance, and must then be prohibited. The diet must be generous and consist chiefly of fish, meat, poultry, milk, and eggs, with a very moderate allowance of wine. The bowels, kidneys, and skin should be kept acting regularly, and a warm bath daily is beneficial. The hours of work must not be excessive, and the patient should retire to bed early and have at least eight hours' sleep. Outdoor exercise is beneficial, but it should never induce fatigue. The clothing must be

warm and cold should be carefully avoided, as patients taking mercury are sometimes very susceptible to its influence. It is absolutely necessary to impress upon the patient the fact that although, under treatment, his symptoms will rapidly clear up, and that he will perhaps never show any others, yet that he must still continue under treatment and observation; if it is found necessary to temporarily intermit the mercurial course, he should further be warned that he must not be surprised or alarmed if a slight eruption makes its appearance during the intermission, and that such eruption will never be serious and will rapidly disappear when the treatment is resumed. At the present day syphilis is popularly regarded with a degree of fear and dread which, under proper treatment, is not justified by results; this should be thoroughly explained to the patient, who should be encouraged and reassured as to his ultimate complete recovery, otherwise his natural anxiety may develop a condition of extreme mental dejection, with perhaps that state which is almost as serious to the patient as is syphilis itself—syphilophobia.

Mercury.—When a mercurial course is entered upon the surgeon should satisfy himself that the teeth are sound, and if any are decayed a dentist should be consulted. In whatever way mercury is administered, its action must be supervised, so that on the appearance of any symptoms indicative of an excessive dose, the drug may be stopped and appropriate measures adopted.

No one preparation of mercury is applicable to all persons, or to all syphilitic manifestations, and if one prescription does not suit the patient some other must be tried; with a little care a preparation can usually be found which is well tolerated and efficient. There is no occasion to push the drug until the gums are tender, as has been advised by some.

Mercurial salivation is easily produced in some persons, with difficulty in others. Salivation is characterised by swelling and tenderness of the gums and the appearance of a blue line at their margins; the salivary glands are swollen and tender and the saliva is increased in quantity; griping pains in the abdomen, with purging, are common. In bad cases the gums may slough, the teeth loosen and drop out, and the alveolar borders of the jaws necrose. Mercurial ulcers must be diagnosed from syphilitic; they may affect the gums, tongue, cheeks, and lips, are accompanied by considerable swelling and pain, and are surrounded by a red inflamed area. The breath has a peculiarly offensive odour.

If mercurial salivation is induced, the mercury must be stopped and a saline aperient ordered. The mouth should be frequently

rinsed with an astringent lotion of alum or chlorate of potash (gr. 10 ad ℥i.), with glycerine and rose water. If the dose of mercury employed is likely to cause spongy gums or yet more serious results, great care must be paid in thoroughly cleansing the teeth twice daily, and the patient may be directed to rinse out his mouth with the alum or potash lotion three or four times a day. A very useful lotion for this purpose is one of trisulphate of alumina, gr. 5; acetate of lead, gr. 2; aqua floris aurantii, ℥i.

Mercury may be administered by the mouth, by inunction, by fumigation, or by hypodermic or intra-venous injection.

Mercury by the mouth is the most convenient method, and is usually given in the form of pills and in combination with opium or Dover's powder to prevent purging. Grey powder, blue pill, or the tannate of mercury in one-grain doses, with an equal quantity of Dover's powder and extract of gentian may be given thrice daily after meals, or more often if a larger dose seems necessary. If the patient is weak and feeble, quinine, iron, or strychnia may be combined with the pill. One of these forms of mercury will usually be well tolerated by most patients; but should salivation, griping pains, diarrhœa, or intestinal disturbance be excited, the amount of Dover's powder must be increased, or pure opium, gr. $\frac{1}{3}$-$\frac{1}{4}$, substituted; pepsin or extract of lettuce are also useful.

If none of these preparations are tolerated, or if they do not produce the desired result, some other form must be prescribed or the dose decreased. The success of treatment and the comfort to the patient during it are materially influenced by the discovery of that preparation and that dose which can be taken without inconvenience; if the patient proves intolerant, more harm than good may result.

The perchloride ($\frac{1}{32}$-$\frac{1}{8}$ grain), combined with gr. 3-4 of iodide of potassium, is a good preparation and well borne by most people. It is best given in a fluid form, as there is considerable difficulty in accurately dispensing iodide of potassium in pills. The green and red iodides of mercury, Donovan's solution, or Plummer's pill often prove most effectual in persistent and relapsing skin lesions.

The iodides of mercury must be combined with opium, and as they are very liable to decompose, the pills should not be kept too long. The green iodide is preferable to the red, as it is less irritating.

Mercury by inunction is useful in cases where the patient

proves intolerant of it by the mouth, or when it is necessary to bring him rapidly under its influence. It has the disadvantage of being a troublesome and dirty method, and of sometimes producing considerable irritation and eczema of the skin, especially if the oleate (5 or 10 per cent) is used.

The blue ointment, mixed with an equal part of lanolin, is the best preparation, and one or two drachms of the mixture should be used at each inunction, which is best made into the loins, groins, axillæ, or upper and inner aspect of the thighs. These situations should be selected alternately, so that irritation may not be produced. Inunction is best made by a skilled rubber, but can be conducted by the patient himself. The part selected should be carefully cleaned, and it is advisable that the patient should have a warm bath before inunction, which should be made at night just before going to bed. The ointment must be gently rubbed in for about a quarter of an hour, in front of a fire if possible; the part is then covered with flannel, and the patient should retire to rest. Inunction should be repeated daily for three periods of six weeks each, with one month's rest between. The gums must be carefully watched, and the alum, potash, or alumina mouth-wash should be freely used to avoid salivation.

During the treatment the patient must be warmly clad and avoid cold; the diet must be liberal and generous, and cod-liver oil and tonics should be given. The Aix-la-Chapelle treatment practically consists in mercurial inunction after sulphur baths, combined with the observance of strict dietary and hygienic rules. During treatment by inunction the patient must be kept under strict supervision.

Mercury by fumigation is especially useful when it is necessary to push the drug and bring the patient rapidly under its influence, and when there is a persistent skin eruption. Lees's bath is the most convenient form, and from 20-30 grains of calomel should be used at each sitting, which must take place at night and not last more than a quarter of an hour. The patient, stripped and sitting on a cane-bottomed chair, is surrounded by a cane-hoop frame, with a woollen cover which is tied round the neck and covered with a blanket, Lees's lamp having a little water in the saucer at the top, and the calomel on the disc is put beneath the chair and lighted. In a few minutes the patient is bathed in perspiration, and the calomel volatilises and is deposited on the skin. When the bath is over, he should be wrapped in a warm blanket and placed in bed by an attendant, who should always be present in

case faintness is induced. Strong and vigorous patients may have a bath daily, but the weakly and feeble should not have one more than every third day. Treatment by fumigation rarely causes salivation but is very depressing, and during it the patient must be very careful to follow the general rules previously laid down; he must avoid cold, take moderate exercise, and live well. He should avoid being out in damp weather and after sun-down.

Mercury by intra-muscular or intra-venous injection is but little used in this country, although advocated by some. It should be reserved for those rare cases in which all other means of treatment prove ineffectual.

A variety of preparations have been used for intra-muscular injection, but the superiority of any one does not appear to be great.

Either of the following preparations may be employed; the sozoiodal is said to be less irritating than the sal alembroth:—

℞ Hydrarg. perchlor. grs. 32.
Ammon. chloride, grs. 16.
Aq. distill. ℥ii.
Ten minims (= ⅓ gr. sal alembroth) for each injection.

℞ Hydrarg. sozoiodal, grs. 5.
Sodii iodid. grs. 10.
Aq. distill. ♏ 200.
Ten to fifteen minims for each injection.

The injections may be made (under aseptic precautions) each day or at longer intervals, according to the strength of the patient and the effect produced. If used daily, the course should extend over about a month. Perhaps the most satisfactory method is to give an injection once a week for two months, then once a fortnight for three months, followed by monthly injections until the end of three years from the date of injection.

The outer part of the thigh, the loin, or buttock are the most suitable seats, and the injection must be made deeply into the muscles; it often causes considerable pain, but suppuration is rare. In some cases symptoms of mercurial poisoning of more or less gravity ensue. This method of treatment is not to be recommended, nor is there reason to suppose that it is more efficacious than when the drug is taken by the mouth. The intra-venous method seems still less to be advisable.

Duration of a mercurial course.—The course of treatment

should extend over two years in all cases, and longer if the disease is severe and the symptoms persistent. To obtain the full benefit of mercury, it must be given in small doses over a long period of time.

The plan I usually adopt is to administer mercury continuously for the first nine months, only remitting it for a few days if the gums become tender. During the tenth month the mercury is discontinued, and cod-liver oil, Fellows' syrup, or any suitable tonic given; during the eleventh and twelfth months treatment is resumed. During the second year the patient should take mercury in smaller doses every alternate month, and during the third, fourth, and fifth years I usually recommend him to take a mixture of the perchloride with iodide of potassium for a month. If this course be followed, and the mercury is given in a form suitable to the patient, it can be well borne and, in most cases, will prove successful. If the patient suffers severely from the disease, treatment must be continued over a longer period.

The iodides of potassium, sodium, and ammonium are specially useful in the tertiary stage, and may be advantageously given with small doses of the perchloride of mercury. All iodides are depressing, and some people are peculiarly intolerant of them, symptoms of poisoning (iodism) appearing after very small doses.

Iodism is characterised by coryza and lacrymation, dryness of the throat and fauces, frontal headache, and increase in the nasal mucus, with the usual signs of an ordinary cold in the head. There is often considerable mental depression and bodily lassitude. Acne and various skin eruptions, sometimes severe and extensive, may be produced, and should such signs appear the dose must be diminished.

It is surprising how much idiosyncrasy influences the action of iodides, some patients responding well to quite small doses, while others require the drug to be steadily pushed as tolerance is established; iodides should be combined with carbonate of ammonia and a vegetable bitter to counteract, in some measure, their depressing effects.

During the secondary stage iodides are often useful, especially if the symptoms do not yield to mercury. Ten grains at night is the most useful and least depressing method of administration.

Serum treatment.—Recently, blood-serum of syphilitic patients has been used hypodermically as a remedial agent. Streptococcus serum has also been used, and Rudolph reports two cases in which accidental erysipelas, occurring in a syphilitic patient, was attended

by rapid and beneficial results; the erysipelas apparently having in one such case more potent curative effects than had mercurial treatment. At present no definite statements on this method of treatment can be made.

SPECIAL TREATMENT OF SYPHILITIC LESIONS

The primary lesion, when non-ulcerating and occurring as a papule, needs no dressing, but should be covered up to prevent irritation. The ulcerated sore must be kept clean and covered with a piece of lint saturated with black wash, or it may be dusted with calomel. If phagedænic symptoms appear, the treatment must be more energetic (*vide* p. 118). If the sore be concealed by a tight foreskin, this should be slit up. Excision of the sore is useless, infection of the blood having already occurred.

Non-ulcerated skin affections do not usually require any local treatment, but if they are very persistent, as in palmar psoriasis, a little mercury ointment may be rubbed into the patches, or they may be covered with Unna's mercurial plaster. Internally, the green iodide of mercury or Donovan's solution are most useful, and may be well combined with the iodide of potassium. Widespread and persistent eruptions may require treatment by fumigation.

Ulcers of the skin must be kept scrupulously clean and all scabs and crusts removed; usually a simple unirritating lotion, such as boracic acid, is all that is needed locally, healing being dependent on the administration of anti-syphilitic remedies. In persistent cases, healing may be hastened by the application of mild mercurial ointments. If the lesions inflame and tend to spread, the acid nitrate of mercury will arrest the process.

Condylomata and mucous tubercles must be kept clean and as dry as possible. They should be dusted with equal parts of calomel and starch powder or zinc oxide twice a day after being washed and dried. When situated in contact with a skin surface (*e.g.* fold of the nates), condylomata should be covered with lint so as to prevent auto-inoculation.

Lesions of the mucous membranes usually heal rapidly if all sources of irritation be removed and a mercurial lotion applied. In the case of the throat, mouth, and tongue, smoking should be prohibited and the patient warned against hot drinks, mustard, sauces, and similar things likely to cause irritation. Astringent and mercurial gargles and mouth washes should be prescribed. Alum or chlorate of potash (gr. 10 ad ℥i.), with glycerine and rose water, or

perchloride of mercury (gr. $\frac{1}{4}$ to gr. 1) are the best gargles. If the throat is very bad and does not readily yield to this, the mercury must be increased to two grains to the ounce. In some cases it is necessary to use very strong solutions (hyd. perchlor., gr. 4, or hyd. cyanide, gr. 2), and these had better be applied with a camel's-hair brush by the surgeon. The patient should always be warned of the poisonous nature of mercurial gargles.

Secondary ulcers of the tongue and cheeks are sometimes very persistent and painful, and if the above lotions do not produce a cure, the ulcers may be touched with nitrate of silver, or daily painted with a solution of twenty grains of the salt to the ounce of glycerine and water. Chromic acid in 10 per cent solution, or crystals of salicylate of soda are also useful local applications; under the former these ulcers often heal with great rapidity.

CONGENITAL OR INHERITED SYPHILIS

Transmission of syphilis. — Syphilis may be inherited through one or both parents. In the latter case inheritance is more certain and the disease more severe. Inheritance through the father alone renders the mother syphilitic through the fœtus; although she may be in apparent health and not show traces of syphilis on superficial examination, yet a searching inquiry and examination will often prove that infection has occurred; and even if no such evidence be forthcoming, the fact that she has been inoculated and thus protected is definitely proved, since she is, with very rare exceptions, immune (Colles's Law). The mother may be infected before or after conception; in the latter case the child may escape, provided gestation is advanced, and usually does so if the mother is not inoculated before the seventh month.

Syphilis is transmissible to the offspring during the primary and secondary stages, but not in the tertiary. The more recent and severe the infection, the more certainly and severely will the child suffer, its chances of escape increasing with the remoteness of infection of the parents; thus a recently infected woman will, if she conceive, abort; in later conceptions abortion may yet occur, but at a later period of gestation; later still, a living syphilitic child is born and subsequent children suffer less severely or escape. To put it shortly, the chances of infection of the child and the severity of that infection diminish with time.

The transmissive power in a woman usually lasts during the first four years of the disease. Transmission to the second genera-

tion does not occur. Hereditary syphilitics are usually immune to the acquired disease; but this immunity is not invariable, nor should we expect it to be so in view of the fact that second infection of acquired syphilis, although rare, is possible.

Abortion in acquired syphilis. — Careful treatment of the mother during her pregnancy may prevent abortion, and the child, although it very rarely escapes altogether in cases of recent infection, may suffer but little. Abortion may be due to disease of the placenta and occlusion of the vessels, to disease of the uterine mucous membrane, or to the general constitutional disturbance attending the onset of the secondary stage. Grave syphilitic disease of the fœtus occasions its death and consequent expulsion.

Course and symptoms of congenital syphilis.—The symptoms differ only in degree and not in essential characters from those met with in acquired syphilis, but since they attack rapidly-growing tissues instead of matured ones, their effects are more serious and lasting. The main stress of the disease falls upon the skin and bones, but in very bad cases the viscera are affected. One noticeable feature of congenital syphilis is the occurrence of secondary and tertiary lesions at the same time. The symptoms occur at an **early** and a **late** stage, the former comprising the first few weeks of life, the latter childhood and puberty. Hutchinson and others assert that the early stage may be absent, but this is very doubtful. These stages are usually separated by a period of apparent health. They correspond with the secondary and tertiary stages of the acquired disease, but the latter shows certain peculiarities in the inherited form (see p. 205).

The early stage.—The primary sore is, of course, absent; should one be detected, the case is one of acquired not congenital syphilis. In cases of recent infection of the mother, the child may be born with syphilitic lesions and usually soon dies; more frequently the symptoms appear within the first six weeks, but the later they manifest themselves the less serious will they be. At birth the child may be apparently healthy and fat, but very soon it shows general deterioration of the health and becomes pale, anæmic, and emaciated; the skin loses its softness, is shrivelled, wasted, wrinkled, and of a muddy tint, and the child has a prematurely aged appearance. Iritis is occasionally present.

During the evolution of this stage the child not infrequently succumbs, the end being ushered in by severe and increasing anæmia.

Affections of the skin and mucous membranes.—Macular

and papular rashes of the skin and mucous membranes are, as in the acquired disease, early and common manifestations. Coppery papules usually occur in groups, and chiefly affect the lower half of the body, especially the buttocks, nates, genitals, and groins; in which situations, as they are kept moist and warm, the papules appear as mucous tubercles. Mucous tubercles also affect the lips, cheeks, tongue, and throat. A most characteristic and early sign is the inflammation and formation of mucous patches and ulcers on the nasal mucous membrane, which is considerably swollen and encrusted with thick yellow scabs, and secretes a copious, tenacious, muco-purulent material. The nasal mischief interferes with respiration and causes "snuffles"; the respiratory difficulty renders sucking difficult, and hence the child is often fretful and restless, and its nutrition further impaired; moreover, cold is often contracted, and may terminate in bronchitis or broncho-pneumonia. The inflammatory affection of the mucous membrane may extend to and cause necrosis of the bones of the nasal fossæ, or their development may be so impaired that the bridge of the nose is permanently depressed. As in the acquired disease, the mucous membrane of the larynx is often affected and the child's cry is harsh and hoarse.

Mucous tubercles at the angles of the mouth or round the nostrils may, owing to the mobility of the parts, lead to troublesome fissuring; when these fissures heal, fine white radiating scars are left. Mucous tubercles of the mouth and tongue are common, and cause pain during sucking; stomatitis—not to be mistaken for aphthæ—may give considerable trouble and lead to sloughing of the gums, to the detriment of the primary teeth, which are often discoloured, decay early, and crumble away.

The vesicular and pustular syphilides may be present at birth, or occur within a few days; they indicate a severe form of the disease, and are of serious omen.

Syphilitic pemphigus (Fig. 37, p. 183) affects the soles and palms, and spreads up the limbs to the trunk. Large bullæ appear situated on dark red papules; they soon suppurate, burst, and scab over, ulceration spreading beneath the scabs. Non-syphilitic pemphigus does not affect the soles or palms.

The nodular syphilides and subcutaneous gummata do not usually occur until the late period of hereditary syphilis, and are always rare.

Affections of the hair and nails are similar to those met with in the acquired disease (p. 185).

Affections of the lymphatic glands.—Coincident with the

appearance of the skin lesions, the lymphatic glands in various parts of the body may, as in acquired syphilis, be enlarged and tender; sometimes the skin over them is inflamed and ulcerates, the exposed glands disintegrate, and the process closely simulates tubercle.

Affections of the viscera are very serious, and usually appear early, or may be present at birth. Interstitial inflammation, gummata, and amyloid degeneration are the morbid conditions, the liver and spleen being their usual site; but no organ is exempt. The affected viscus may be found to be enlarged. Anasarca, with purpuric spots, is an occasional associated condition.

Affections of the bones and joints may be early or late, and may implicate the flat bones of the skull or the periosteum or epiphysary lines of the long bones, especially those of the forearm and leg. Any of the bone or joint lesions met with in acquired syphilis may occur in the inherited disease, but two forms are to be specially mentioned: (1) epiphysitis or osteo-chondritis with pseudo-paralysis, and (2) chronic effusion into one or more joints. When the disease affects the periosteum, inflammation is excited, and layers of soft, white, porous new bone are deposited on, and parallel with, the long axis of the shaft, from which they are clearly demarcated. Sometimes this new tissue is more highly vascular and fibrous, resembling that formed in rickets (rachitic or spongioid form), or the two forms may be combined. As these periosteal changes advance, the normal shaft undergoes decalcification, and hence spontaneous fracture or bending is not uncommon. The deposit of new bone may lead to considerable thickening of the shaft.

The changes in the epiphysary line closely resemble those met with in rickets; thus there is increased cellular proliferation but imperfect ossification, and the cells may soften and form gelatinous areas like fruit jelly (gelatiniform atrophy). The end of the bone is considerably enlarged, through the deposit of new tissue from the periosteum. The epiphysis may be completely separated and suppuration may occur, although this is rare. Occasionally the joint is involved. The interference with the process of growth and development leaves the bones short and stunted. When the bones are very soft—and especially if they are broken—the limb hangs uselessly and appears as if paralysed (pseudo-paralysis). When the flat bones of the skull are affected the periosteum is usually involved, but gelatiniform atrophy is not so common.

Parrot's bossing and craniotabes are thought by some to be essentially syphilitic in nature, while others consider them as of

rickety origin. It is probable that both diseases (and they are often associated) may induce these changes, and that craniotabes at least may be dependent on any condition of general ill-health impairing nutrition. Parrot's bossing and craniotabes may be met with in the same skull.

Parrot's bossing is dependent on inflammation of the periosteum, and consequent heaping up of soft, spongy, highly vascular new bone of a deep maroon colour. The new bone is composed of large medullary spaces, with perpendicular trabeculæ irregularly arranged round the Haversian canals; when the active stage has passed the density increases. The bossing is remarkably symmetrical (Fig. 41), is present on the outer table only, and follows more or less definite lines. It especially affects the frontals, parietals, and peribregmatic area, at the same time respecting the frontal, parietal, and occipital eminences, which afford a striking contrast to the affected parts. The sutures may be encroached upon and obliterated, so that the full development of the skull is impaired. The fontanelles may close prematurely or may remain widely open, islands of new bone being present in them. The frontal bosses broaden the skull and give it the peculiar shape and greater breadth so often seen in the subjects of hereditary syphilis (natiform skull).

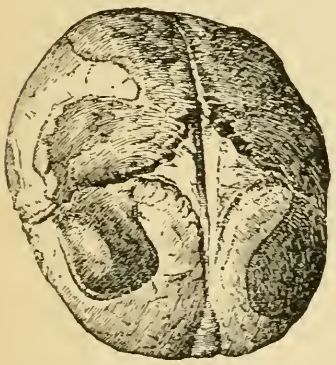

FIG. 41. — Parrot's bossing of the skull in a congenitally syphilitic child. The symmetry of the change is well shown. The anterior fontanelle is widely open and the two halves of the frontal bone are separate. (Westminster Hospital Museum, No. 21B. Drawn by C. H. Freeman.)

Craniotabes is symmetrical, but is often more marked on one side, usually the right, and especially affects the postero-inferior parietal angle. The inner table of the bone is atrophied, and the small depressions produced are exaggerations of those corresponding with the cerebral convolutions. Atrophy leads to thinning, and small conical pits are seen on the inner surface of the skull; there may be a fine lacework of bony tissue, or else actual perforation, the dura mater and periosteum then being in contact. Numerous patches of atrophy may be present, and can be detected during life as weak spots yielding to the pressure of the finger. The atrophy proceeding from within does not cause bulging externally, since the skull is supported by the pillow; it is probable that the atrophy is induced by the pressure of the brain on predisposed bones.

Craniotabes is rarely present before the third month of life, and all traces of it have usually disappeared by the twelfth. Parrot describes cases of congenital craniotabes presenting characters different to the infantile form. In these the affection occurs along the margins of the sagittal suture and round the bregma, and small, dome-like projections, often perforated at the summit, are present externally. These are supposed to be due to the absence of external support counteracting the pressure of the brain within; which pressure is exerted on the vertex of the skull, since the head, *in utero*, is usually the most dependent part.

The late stage.—The symptoms of this stage are in many respects similar to those of the tertiary period in acquired syphilis, but Hutchinson is doubtful whether they ought not to be regarded as secondary, to which, in some respects at least, they have a greater resemblance. Thus the lesions are usually symmetrical, and tend to spontaneous cure rather than to persist and recur. If these symptoms are regarded as belonging to the true secondary stage, it must be admitted that the inherited disease has no tertiary period. Symptoms may appear at any time, but usually do so at the period of second dentition or puberty, or later still, during the final stages of development of the skeleton. The later the symptoms appear the better, since delay in their manifestation argues a more favourable course, and they are less likely to do serious damage to tissues more mature. Sometimes the late stage follows closely on the early symptoms, in other cases it may never appear. The lesions of the late stage clinically resemble those of tubercle, and the diagnosis often depends on the evidence of previous syphilitic mischief, the general appearance of the child, and the effects of treatment. The appearance of a syphilitic child may or may not be characteristic. He is, in consequence of the early affections of the bones, often stunted and dwarfed, the forehead is square and prominent, the bridge of the nose depressed, and the child is generally ill-developed. Fine white linear scars (the remains of fissures and mucous tubercles) are not uncommonly seen at the angles of the mouth or round the alæ nasi. The long bones may show unmistakable signs of periosteal or epiphysary disease, but it must be remembered that these signs are very like the changes of rickets. Three very characteristic features of inherited syphilis are interstitial keratitis, malformation of the upper central incisor teeth, and symmetrical deafness, unattended or preceded by discharge (Hutchinson's triad).

Affections of the skin in the late stage are not common, but

syphilitic "lupus" and gummatous ulcers of the legs may be present. Chronic osteitis and periostitis, caries, and necrosis are not uncommon.

Chronic painless effusion may take place into a joint, usually the knee, accompanied by thickening of the synovial membrane. This condition may persist for weeks or months, but gradually clears up under treatment. More than one joint may be affected.

Deafness is by no means uncommon, and usually appears between the ages of ten and fifteen; it is dependent on changes in the internal ear.

Affections of the teeth.—The milk teeth usually escape, but may decay early and crumble away if there has been severe stomatitis.

The permanent teeth may or may not be affected. The upper central incisors are the ones showing characteristic changes, but all may be badly and irregularly developed, and more or less pitted if mercury has been injudiciously pushed. The central incisors are small and peg-shaped, being broader at the base than at the cutting edge, which latter crumbles away in the centre so that a crescentic notch is formed, and the dentine is exposed by removal of the enamel (Fig. 42).

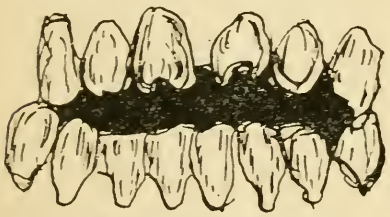

FIG. 42.—Malformation of the upper central incisor teeth in inherited syphilis. (Hutchinson, Allbutt's *System of Medicine*.)

Affections of the eye.—Interstitial keratitis is in the great majority of cases due to inherited syphilis, but is occasionally seen in the acquired disease.

It nearly always begins between eight and fifteen years of age, first in one eye, and within a few weeks in the other. In the early stages there may be some ciliary congestion with photophobia, and the disease is considered as secondary to inflammation of the ciliary zone. The cornea becomes hazy, loses its lustre, and is eventually rendered quite opaque, and resembles ground glass. The change usually begins about the centre, but the margins are also affected. If the disease is severe and persistent, leashes of vessels shoot into the cornea from the margin; this may be limited to one segment (vascular keratitis). Ulceration of the cornea very easily occurs, and the inflammation may extend to the ciliary body or to the iris. After some weeks or months the eye first affected begins to clear up, and in the course of time (often many months) the opacity may almost entirely disappear, although the cornea will never regain its

normal lustre. Under prompt and efficient treatment the prognosis is usually good.

Treatment.—If during the early stages there is much photophobia, the child should be kept in a dark room or wear a shade, and eserine or cocaine must be dropped into the eyes. Constitutional treatment is of course necessary. To promote absorption a little yellow oxide of mercury ointment should be put into the eyes every day.

Affections of the viscera are similar to those met with in the acquired disease. They only occur in bad cases.

Prognosis.—The more recent the infection of the parents, and the earlier the symptoms appear in the child, the more grave is the prognosis. In some cases the disease may give no trouble after the first few months; in others, symptoms recur during all the years of growth, and lead to serious consequences. Visceral lesions are especially grave. During the early stage the affection of the nasal and respiratory mucous membrane not only enfeebles the general health, but lays the child open to bronchitic and pulmonary mischief.

Syphilitic children are less able to combat the ordinary maladies of childhood than are healthy ones.

Treatment.—The early lesions of inherited syphilis are contagious, and every care must be taken that the child does not inoculate its attendants. The mother, who is immune, should nurse her own child; if she is unable to do so, it must be brought up by hand, and on no account should a wet nurse be employed. Syphilitic infants require the utmost care and attention; they must be carefully fed, properly clothed, and prevented from catching cold. No special dietary is indicated, but some advise the use of goats' or asses' milk in preference to that of the cow.

Cod-liver oil, maltine, and the syrup of the phosphate or iodide of iron are advisable, if they can be taken without causing gastric disturbance. Mercury is to be administered by the mouth or by inunction; the latter method is the most convenient, and is less liable to induce intestinal trouble. The child should have a warm bath in front of a fire; when thoroughly dry, a piece of linen spread with about half a drachm of mercurial ointment (which may be mixed with an equal quantity of lanolin) is placed over the abdomen or round one of the thighs, and retained in position by a flannel roller. The seat of inunction should be frequently changed, so as to avoid irritation of the skin. Care must be taken that salivation is not produced, and the inunction should be occasionally remitted.

If mercury is to be given by the mouth, half a grain of gray powder, with a little sugar, should be given thrice daily, and the dose may be increased or diminished according to the effects produced. Constitutional treatment, with intervals of rest, should be maintained for at least six months, and longer if the symptoms indicate it.

Ulcerations about the nasal and buccal mucous surfaces should be kept clean; all crusts should be removed from the nose, and the discharge cleared away with a camel's-hair brush; the surface may then be painted with a weak solution of the perchloride of mercury with borax. Mucous tubercles about the genitals, anus, etc. must be kept clean and dry, and dusted with calomel and starch powder.

The iodides of potassium, sodium, and ammonia are useful, in combination with mercury, in the later stages, and may sometimes be used with advantage should the earlier symptoms prove resistant to mercury alone.

Disease of the bones leading to caries or necrosis will require local surgical treatment in addition to the constitutional.

SOFT CHANCRE OR CHANCROID

Etiology.—Soft chancre is a highly contagious, local, venereal ulcer, never leading to general infection of the blood, but often accompanied by bubo. One attack does not confer immunity against others. Chancroid is due to Ducrey's bacillus, which is small and short, with rounded ends, and stains deeply with gentian-violet, but not by Gram's method; the central portion of the bacillus remains clear. This bacillus is often arranged in chains. It has not yet been cultivated, but experimental inoculation practically proves its specific qualities. It is not met with in any other form of ulceration, although other organisms—especially the ordinary pyogenic varieties—are present with it in soft sores. The organism is very virulent, but is destroyed by drying; it gains entrance through abrasions of the skin or mucous membrane, but does not produce any ill effects on these when uninjured. Ducrey's bacillus is sometimes found in the pus of the bubo accompanying soft chancre; in such cases the pus, if inoculated, is capable of producing a typical soft sore; in most cases, however, the suppuration is due to the pyogenic organisms associated with soft chancre, and is then not capable of producing a chancre, nor does the bubo show such virulent, inflammatory, and destructive tendencies. It is probable that in the case of acute or chronic inflammation of the glands

without suppuration, the process is due to absorption of the toxines of the organisms by the lymphatics, rather than to the spread of the microbes themselves.

Direct inoculation during sexual intercourse is the rule, but mediate contagion by fingers, towels, etc. may occur. Auto-inoculation is not uncommon, and is usually due to scratching.

Situation of the sores.—Soft chancres are nearly always multiple, and are situated on the genitals. In men, the furrow behind the glans penis, especially near the frenum, is the most usual seat of the lesions; sometimes they are scattered over the glans, and there is much associated balano-posthitis, especially in those of uncleanly habits.

The skin of the penis and scrotum is rarely affected, but if the prepuce is long and tight, so as to be liable to fissuring during intercourse, numerous soft chancres may be present at its free margin. In women, the entrance to the vagina, the fourchette, and the mucous surface of the labia are the favourite seats; the vagina higher up is rarely affected. Sometimes soft chancres are present round the anus, either from auto-inoculation or from unnatural intercourse; the latter mode of infection may be suspected when the sores are placed close to and immediately round the anus.

Clinical characters of chancroid. — Chancroid has no appreciable period of incubation, the pathological process beginning immediately after inoculation, as has been demonstrated by numerous experimental inoculations. The patient's attention may not, however, be drawn to the sores for a few days, especially in the case of women. At the seat of inoculation a small red pimple, surrounded by a narrow zone of acute inflammation, makes its appearance. Suppuration ensues within forty-eight hours, and a pustule, seated on a red inflamed base, results; the pustule bursts, leaving a characteristic ulcer.

The ulcer is shallow and clean cut, as if punched out; its base is spongy, and covered with a greenish-yellow purulent exudation, but there is no induration unless the sore has been irritated. Such a sore is very painful, and is usually quite small unless adjacent ulcers have fused, or inflammation has been excited.

The ulcerative process usually extends for a few days, and numerous sores may be present; when spreading has ceased the ulcers remain stationary for some days, and then granulate, healing being complete in from three to four weeks from the onset; the site of the sore is then indicated by a small scar.

Complications.—Inflammation.—If irritated by neglect or dirt,

or by injudicious treatment, soft sores may become acutely inflamed, and the ulceration may spread rapidly and suppurate freely. In such cases inflammatory induration may occur at the base, so that the ulcer simulates a hard chancre, but the induration is not so dense nor is it so distinctly circumscribed; moreover, in the case of hard chancre, pain and acute inflammatory signs are absent.

Balano-posthitis is by no means uncommon, especially if the prepuce is long and tight, and the patient regardless of personal cleanliness. When present, it causes acute inflammatory phimosis, with much œdema and profuse discharge, and may even result in gangrene of the prepuce.

Phagedæna may attack soft sores, especially if they are concealed beneath a tight prepuce.

Bubo.—Soft chancre shows a marked tendency to involve the lymphatic vessels and neighbouring glands, which frequently suppurate—more often, however, in men than women. Sometimes the glands escape completely; in other cases they inflame but do not suppurate, or the acute inflammation may subside, leaving the glands chronically enlarged, and slow suppuration follows. In the last class of cases, the sore, having completely healed, may remain undetected, and this has led some to think that the venereal poison may be absorbed by the lymphatics and conveyed to the glands without any local lesion occurring at the point of inoculation (*bubon d'emblée*). There is no genuine evidence in support of this view, and the mere denial of a sore on the part of the patient will, of course, be accepted with due caution.

The glands affected are those along Poupart's ligament—the superficial inguinal; but in some cases the deep glands in Scarpa's triangle are implicated, and in one case under my care those along the external iliac artery suppurated. Both sides are often affected, especially if the sores are near the frenum.

The glandular involvement usually begins about the end of the second week, but may be earlier or later. The affected glands are swollen and acutely tender, and when suppuration occurs the cellular tissue and skin become œdematous, and the latter is red and thinned as the pus approaches the surface. Unless the abscess cavity be freely sharp-spooned, the destructive process may continue to spread and large areas of tissue be destroyed; sinuses may burrow a long way, and the patient may be placed in considerable danger if the femoral trunk or its branches are encroached upon, as secondary hæmorrhage may ensue.

When the glands inflame, constitutional symptoms with slight fever may occur, and are considerably aggravated with the advent of suppuration.

Diagnosis.—Soft chancre may be distinguished from hard by the absence of an incubative stage and of induration, by the multiplicity of the lesions and the evident ulceration accompanied by pain, by the acute lymphadenitis frequently terminating in suppuration, and by the absence of succeeding secondary symptoms. An inflamed soft chancre only superficially resembles a hard one, and a little care will usually avoid any mistake. It must, however, be remembered that a soft chancre may also be inoculated with the poison of syphilis, and will then indurate when the incubative stage of that disease has expired (mixed chancre).

Herpes usually appears as a crop of vesicles accompanied by considerable itching and irritation, but no pain or glandular enlargement. The vesicles dry up, and do not usually ulcerate if they are kept clean and dry. Repeated attacks are common.

Treatment.—If there is phimosis, the prepuce should be slit up so that the sores may be fully exposed. Circumcision may be performed at once, provided the sores are so situated that the raw surface can be protected against inoculation; if this cannot be done, or if there is inflammatory phimosis, the operation had better be postponed until the morbid process is cured.

The parts must be kept clean by washing and bathing with boracic acid lotion (gr. 4 ad ℥i.), the glans should then be dried carefully, and the sores lightly dusted with crystals of iodoform, the unpleasant smell being minimised by the admixture of coumarine or coffee. The dressing should be repeated at least twice daily, and if the sores are so situated that auto-inoculation is likely to occur, they should be covered with a small piece of boracic lint. Iodoform does not invariably effect healing, and when it fails black-wash or calomel may be substituted.

Some surgeons prefer to destroy the ulcer at once with caustic, in the hope of averting glandular abscess. This may be done, after thoroughly drying the parts, by touching the ulcers with pure carbolic acid, sulphate of copper, nitric acid, nitrate of silver, or by applying Ricord's paste, which is composed of strong sulphuric acid and willow-charcoal and will be found extremely useful. The pain caused by these applications may be minimised by the previous use of a 20 per cent solution of cocaine.

If phagedæna occurs, the treatment must be prompt and radical (see p. 118).

As soon as the ulcers begin to heal, a simple unirritating dressing, such as boracic acid lotion, is all that is needed.

Treatment of bubo.—As soon as glandular inflammation is present, the patient must be kept quiet to prevent additional irritation. The bowels should be opened and kept acting. Hot fomentations and extract of belladonna with glycerine may prevent suppuration, or will hasten the process if imminent. When an abscess has formed, it should be freely opened by an incision passing obliquely across Poupart's ligament in a direction downwards and inwards. All the diseased gland should be removed with the sharp-spoon, and the resulting wound must be thoroughly cleaned with 1-20 carbolic acid solution and dusted with iodoform. A piece of gauze should then be placed between the edges of the wound, and a dry dressing applied; this may be left untouched for four or five days, when the wound will be found to be granulating healthily. If, however, there is much surrounding inflammation, hot boracic fomentations are more appropriate. If chronically inflamed glands break down, the whole mass is best dissected cleanly away. The resulting wound will usually heal rapidly by first intention.

CHAPTER X

Surgical Infective Diseases (*Continued*)

The General Infective Diseases

By a general infective disease we mean one that is dependent on the invasion of organisms which gain entrance to the blood-stream either directly through the veins or indirectly through the lymphatics. The organisms are capable of multiplying in the body generally, and therefore of producing symptoms out of all proportion to the amount of the original dose. Pathogenic organisms which have gained entrance into the blood may pass into the tissues.

The acuteness and severity of any general infective disease vary with the organism causing it. Some of them, *e.g.* septic infection, are rapidly induced, and run an acute course with a usually fatal termination; others, such as syphilis, run a chronic course, and are amenable to treatment. A third group, *e.g.* tubercle and anthrax, may remain quite local for a longer or shorter period, and general infection may be prevented by timely and radical treatment; but if the local lesions are neglected, such general infection —at any rate in the case of anthrax—will certainly ensue. In some instances an organism, which usually only induces a local infective process, may enter the blood-stream and cause general infection; thus, the gonococcus may excite gonorrhœal rheumatism and be found in the synovial exudation, and, according to some, the streptococcus erysipelatis may behave in the same way (see p. 123).

Inoculation with a general infective disease occurs through a wound; but, as in the case of the local infective diseases, the mere presence of the organism does not necessarily entail the

establishment of the disease, certain favouring causes being essential. Thus in the case of acute necrosis (a form of septic infection or pyæmia) it has been shown that if the staphylococcus pyogenes aureus be injected into the veins of healthy rabbits, the condition is only produced when a bone has been injured, and hence a weak spot formed where the organism can gain the upper hand.

ACUTE SEPTIC INFECTION AND "PYÆMIA"

Etiology.—Septic infection and pyæmia are clinical terms implying an assemblage of pathological and resulting clinical features, due to the action of micro-organisms capable of multiplying in the blood and tissues.

These two states are regarded by many as being distinct, but in reality it appears that so-called pyæmia is merely septic infection accompanied by the formation of secondary centres of suppuration, which are due, not to any inherent difference in the organisms present, but to the longer duration of their action, or to the more favourable conditions under which this occurs.

The ordinary pyogenic organisms are those most commonly met with in septic infection and pyæmia, the most virulent and commonest being the streptococcus pyogenes and staphylococcus pyogenes aureus and albus (Figs. 9 and 10, pp. 40, 41). Any of these may be present alone; but mixed infection is common, and seems to act more virulently.

The general causes favouring the development of septic infection have been already mentioned (p. 104).

The disease can, and ought to be, prevented. Its occurrence in accidental or surgical wounds points to some imperfection in the antiseptic method employed. Foul, ill-drained wounds with small external openings, and those implicating the medullary cavities of bones, joints, veins, and serous cavities, offer every facility for the absorption of the virus and general infection.

A pyæmic process may occur independently of any wound, *e.g.* ulcerative endocarditis and acute necrosis, absorption taking place through the digestive or respiratory mucous tracts. No doubt many of us are thus subjected to the invasion of organisms capable of producing the most serious results; that they do not invariably do so is dependent on the resisting powers of the healthy body, but should this be diminished infection will occur. Septic infection and pyæmia may be acute or chronic.

ACUTE SEPTIC INFECTION (SEPTICÆMIA)

Symptoms.—The symptoms of acute septic infection are similar to, but more severe than, those met with in acute septic intoxication (p. 106). The onset is sudden and ushered in by a severe rigor, and the temperature rises to 104° or 105° F., with nocturnal exacerbations. Headache, nausea, and vomiting are constant, and diarrhœa is by no means an uncommon symptom. The rigor and profuse sweating weaken the patient, and nervous prostration is rapidly induced. The pulse is rapid, and soon becomes feeble and perhaps irregular. The respirations are hurried and shallow, the skin hot and dry, but towards the end is covered with a profuse, clammy sweat. The urine may contain albumen. Delirium at night is the rule; and as the nervous prostration becomes more profound, and typhoidal symptoms show themselves, it deepens into coma before death. Any wound which may be present ceases to heal, and may inflame. The discharges are putrid and offensive.

Death usually occurs within a week from the onset, and is often preceded by purpuric spots about the body.

Diagnosis.—Septic infection may be mistaken for septic intoxication. In the former state, the greater severity of the symptoms, and the fact that they continue after all putrefying material has been removed from the wound, are the diagnostic features.

Post-mortem appearances (see p. 218).

Treatment is of little avail, the great majority of cases proving rapidly fatal. The wound must be thoroughly disinfected and drained. The general treatment consists in the plentiful administration of good, nutritious fluid food, with free alcoholic stimulation. Bark, quinine, and ammonia, with digitalis or strychnine, if the heart fails, are the most useful drugs. If the temperature runs very high large doses of quinine or antipyrin, or cold packing may be resorted to. The action of the kidneys and bowels must be promoted.

Anti-streptococcus serum is employed in cases of infection by streptococci. At present its therapeutic value is undetermined, for in some cases it is apparently very beneficial, while others remain *in statu quo;* it may be generally stated that the remedy if it does no good will not be productive of harm. It should be used as soon as the symptoms are manifestly due to streptococcus infection, and the dose (10 c.cm.) should be repeated about every six hours. In

successful cases the temperature falls, and the local and general symptoms rapidly improve.

CHRONIC SEPTIC INFECTION

The chronic disease is more likely to occur in those who are broken down by some old-standing disease. The symptoms are the same in kind, but differ in severity from those met with in the acute form. Rapid anæmia and emaciation are produced, and the spleen is always enlarged. Death occurs in about half the cases, but the course of the disease may extend over weeks or months. The treatment is similar to that of acute septic infection.

ACUTE SEPTIC INFECTION WITH SECONDARY ABSCESSES (PYÆMIA)

The general course of pyæmia precisely resembles that of septic infection, and the constitutional symptoms are similar, though perhaps even more severe; but there are in addition signs and symptoms dependent on the establishment of secondary suppurative foci.

Symptoms.—Pyæmia is ushered in by a very severe rigor, which commences a few minutes after the temperature begins to rise. During this rigor the lips are blue and cyanosed, and the surface of the skin is pale and cold; the temperature reaches 105°-106° F. As the cold stage passes off the skin is suffused with blood, and the patient is bathed in profuse perspiration; the temperature falls, and he is left very much weakened. In the great majority of cases the rigor is repeated within twenty-four hours, and many others may follow. Each rigor is marked by a fresh elevation of temperature, which again falls during the sweating stage, only to be succeeded by another elevation. The wide fluctuations of temperature within a short space of time are very characteristic of the disease, and although the temperature may fall to the normal or even below it, it never remains low. Between the rigors, when the sweating has subsided, the skin is hot and pungent, and, as the disease progresses, assumes an icteric, sallow tint, with evanescent patches of erythema. The expression is anxious, careworn, and apprehensive; the eyes are bright, or may be leaden and surrounded by dark rings. The cheeks are pale or flushed, especially the latter if the lungs are implicated by secondary inflammation. Herpetic patches may appear about the mouth or alæ of the nose.

Respiration is hurried and shallow, and may be very rapid or

impeded if pneumonia or empyema is present. The breath has a peculiar sickly-sweet smell. The lips and teeth are loaded with sordes, the tongue is dry, brown, and cracked, the mouth clammy; thick, tenacious mucus hangs about the throat and fauces, sometimes occasioning the patient much annoyance. Anorexia, thirst, and nausea are present. The bowels may be confined, or profuse diarrhœa may further increase the prostration. The urine is scanty, high coloured, and probably albuminous. Cardiac failure and nervous prostration are marked. At first there is headache during the day, which disappears as nocturnal delirium comes on; later on, delirium of a low, busy, muttering nature is constant (*typhomania*).

Emaciation and anæmia are rapidly produced, and towards the end the patient falls into the typhoid state. He is lethargic and profoundly indifferent to all around him, delirium and coma may alternate; risus sardonicus, floccitatio, subsultus tendinum, and profuse diarrhœa, with involuntary evacuations of an offensive nature, usher in the end.

The secondary abscesses may not produce any definite symptoms, either because they are so small, or because of their situation in unimportant parts, or, again, because the condition of the patient is so grave that he does not show any evidence of their presence.

The abscesses, which are due to the lodgment of infective emboli, usually appear about five days from the onset of the disease. No organ or tissue of the body is exempt, but certain parts are more particularly affected; the joints, subcutaneous tissue, lungs, liver, kidneys, and spleen are the principal seats of secondary inflammation and suppuration. The occurrence of inflammation may be marked by pain and swelling of the part, or by some symptom referable to the viscus involved. Of the joints, the knee is the one most usually affected, but others are rapidly attacked; if the patient survives long enough, suppuration ensues, and numerous sinuses may form. Subcutaneous abscesses or diffuse patches of cellulitis, which may or may not suppurate, are common.

FIG. 43. — Colonies of micrococci within the hepatic capillaries (Ziegler).

The serous membranes are not infrequently affected; as a rule, their implication is secondary to visceral lesions, but is not necessarily so. The formation of pyæmic abscesses is remarkably rapid; they

may be very numerous and never attain a large size. They contain an oily, watery, and greasy pus.

Diagnosis.—The chief diagnostic features are the repeated and severe rigors and characteristic fluctuations of temperature occurring in a patient with a foul wound. Later on, the secondary inflammations and abscesses and the rapid downward progress of the case render the diagnosis certain.

The joint lesions in acute rheumatic fever make their appearance earlier, and do not lead to suppuration; moreover, the repeated rigors are absent, and the temperature remains continuously high. In acute rheumatism there is a characteristic sour smell in contradistinction to the sickly, earthy smell of pyæmia; moreover, the tongue is covered with a thick, creamy fur in the former condition, and is dry and brown in the latter. Attacks of ague are marked by distinct periodicity, the patient being perfectly well during the intervals; there are no secondary lesions.

Prognosis.—Acute pyæmia is almost always fatal in from four to ten days.

Post-mortem appearances of septic infection and pyæmia.—These are similar to those met with in sapræmia and septic diseases generally. In pyæmia there are the secondary abscesses.

Post-mortem decomposition sets in early and proceeds rapidly, so that the whole body speedily swells from gaseous accumulation, and may be almost unrecognisable. There is marked staining of the dependent parts and tissues generally, and the surface veins are clearly mapped out. The various organs are congested and œdematous, and may be inflamed. In pyæmia infarcts and embolic abscesses are common. The spleen is enlarged, soft, and may be diffluent. The serous cavities contain an increased quantity of fluid, which is turbid and blood-stained. Petechial patches and purulent collections are common. The wound will be found to be in a sloughy condition; the veins leading from it are acutely inflamed and plugged with breaking-down thrombi, which are loaded with micro-organisms. During life these thrombi disintegrate and break down, giving rise to emboli charged with the infective agent. Such emboli, being carried to distant organs and tissues, excite in them at their points of lodgment the secondary abscesses, and thus additional phlebitis and thrombosis are occasioned, and the process is repeated indefinitely. The walls of a pyæmic abscess are often sloughy and ill-defined on account of the rapidity of its formation and the highly irritating

nature of its cause; inflammation often extends a long distance round.

Micro-organisms are found in all the tissues and organs, in the vessels and lymphatics, and in the purulent collections and other fluids.

Treatment.—The treatment of acute pyæmia is conducted on the same lines as for septic infection. Secondary abscesses should be opened and disinfected, provided their situation admits of such a procedure. In some cases general infection may be prevented by ligaturing a thrombosed vein leading from the seat of inoculation (see Otitis Media, chap. xi. vol. iii.).

CHRONIC PYÆMIA

Chronic pyæmia may extend over many months and terminate in death or recovery. The symptoms are similar to, but less severe than, those of the acute disease. Abscesses form in the joints and subcutaneous tissues, but the vital organs are not affected, otherwise death would speedily ensue. Abscess after abscess may form, inflicting much suffering and ultimate damage. Emaciation and anæmia are very marked. The treatment is the same as for the acute disease.

EQUINIA OR GLANDERS—FARCY

Etiology.—Glanders is a common disease among horses, asses, and mules, and may be communicated by them to man, but in spite of its highly contagious nature such communication is rare. Infection may occur through the mucous membrane of the nose and upper respiratory tract or through a wound; in the former case it is probable that in most if not all instances a small scratch or abrasion is present. Glanders is the term usually applied to acute equinia, the chronic forms being known as farcy, but there is no real difference in the morbid processes. The *bacillus mallei* has been proved to be the infective agent. It resembles the B. tuberculosis, but is broader, motile, and stains differently. It is abundantly present in the farcy buds, abscesses, nasal discharge, and in the blood and lymph.

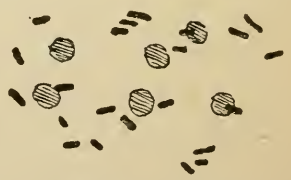

Fig. 44.—Bacillus mallei (glanders) and red blood cells.

Signs and symptoms.—The acute form of glanders is that

most common in man, and symptoms make their appearance within fourteen days of inoculation, and may apparently arise at any date during that period.

The leading characters of glanders are the formation of papules and nodules in the skin, mucous membrane of the nose, lungs, etc., and the formation of deep-seated abscesses; the lymphatics are inflamed and the glands enlarged, especially in chronic cases, and general infective poisoning terminates the case. The onset may be gradual and marked by lassitude and malaise, with general pains of a rheumatic nature; more rarely a rigor ushers in high fever and the characteristic symptoms.

The wound, if there be one, inflames and the lymphatics and adjacent glands participate. In a few days cutaneous tubercles (*farcy buds*) make their appearance and, affecting the mucous lining of the nose, give rise, when ulceration occurs, to an offensive, sanious, watery discharge which later on becomes slimy and tenacious. The disease may spread to the air-sinuses. The tubercles on the skin appear in crops and resemble in some respect the lesions of smallpox. At first they are vesicular or hard and shotty papules situated on a broad, inflamed, and indurated base. Softening and suppuration of the nodules with hæmorrhage into them soon occurs; the pustules break down and give rise to sloughy ulcers which, by confluence, may cover considerable areas. Occasionally the vesicular lesions do not suppurate or rupture, but dry up and scab. Deep abscesses may make their appearance in the limbs, or suppurative synovitis may occur, the patient presenting the usual signs of pyæmia. If the respiratory tract is specially involved, bronchitis or pneumonia supervene.

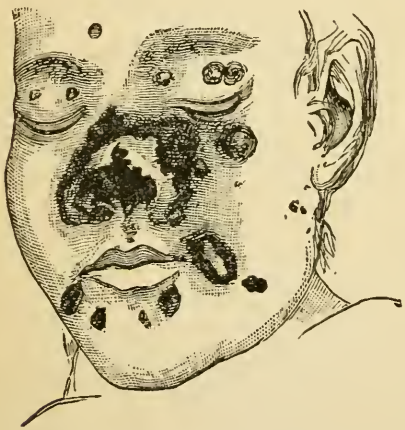

FIG. 45.—Acute glanders eight days after infection (Tillmans, after Birch-Hirschfeld).

The constitutional symptoms are those common to all acute infective processes. The temperature gradually rises, attaining a height of 104°-105° F., and shows marked fluctuations. Constipation is usually present at first, but may be followed by bloody diarrhœa from involvement of the intestinal tract. There is marked cardiac and nervous depression, and the patient, soon passing into

the typhoid state, becomes comatose and dies in from one to three weeks after the onset. In the chronic form (*farcy*) the signs are similar in nature but less marked, and gradual recovery or death from exhaustion may result.

Diagnosis.—When the disease is fully developed the diagnosis is easy, but at the onset glanders may be mistaken for acute rheumatism or septic infection. The diagnosis rests chiefly on the history of the case and known exposure to infection, the characteristic discharge from the nose, the skin lesions, and the usually gradual onset.

In doubtful cases of glanders among horses, the diagnosis can be confirmed by the injection of *mallein*—an extract of pure cultures of the bacillus. If the animal is infected, the injection causes a rise of temperature.

Prognosis.—Acute glanders is fatal, but about 50 per cent recover when the disease is chronic.

Treatment.—A wound which has probably been infected should be completely excised or freely cauterised. The treatment of the disease consists in keeping up the patient's strength with plenty of easily digested food and stimulants, and the administration of quinine. Strychnia, arsenic, and mercurial inunction are specially recommended by some. The ulcers must be treated with strict asepsis, and the nasal douche, coupled with the insufflation of iodoform, should be freely used. Great care must be taken that the attendants are not inoculated by the highly contagious discharges.

It has been suggested to employ mallein as an anti-toxine; it has apparently been beneficial in chronic cases.

CHAPTER XI

Tumours and Cysts

Tumours

Definition.—A tumour is a new growth of simple or complex nature, arising independently of inflammation and serving no useful physiological purpose. The term neoplasm or new growth is not intended to imply that tumours are composed of some tissue normally foreign to the body, since all have their anatomical prototypes. A tumour is, in fact, only a new growth in the sense that the tissue cells of which it is composed grow in an atypical and irregular manner, are often imperfectly developed, and may be present in situations where they are not normally found. Moreover, such new growths either remain more or less distinct from their surroundings or gradually infiltrate and invade them, tending to progressive increase in size and to early degeneration.

A hypertrophy differs from a tumour in that its growth proceeds in a perfectly regular and uniform manner in accordance with recognised physiological laws, in response to some increased need for additional development. The whole organ or part is enlarged, and the overgrown or added elements fulfil a definite physiological purpose, and do not degenerate or produce harmful results.

Inflammatory tissue, such as is seen forming tumour-like masses in the infective granulomata (tubercle, syphilis, actinomycosis, etc.), differs from a tumour in being traceable to a very distinct known cause, and in its tendency to undergo absorption, to organise, or to break down and suppurate.

Etiology.—Very little is known as to the actual causes leading to the growth of tumours; it is most probable that they are com-

plex and variable; and that one form of tumour may be the outcome of factors totally inadequate to produce another.

Heredity.—Hereditary tendency to the growth of tumours is evidenced by the frequent occurrence of growths of the same nature (*e.g.* fatty) in members of the same family, a tendency sometimes traceable through many generations. The inherited tendency is usually local, that is, it affects a special tissue (*e.g.* the epithelium in the case of cancer) and not all tissues alike. It is the predisposition to tumour formation, not the tumour itself, which is hereditary. While we all recognise the influence of heredity we do not thereby explain much; we only accord to tumours what we daily recognise as true of our physical and mental qualities, viz. that a parent may transmit to his offspring certain individual or local characteristics which he himself possesses.

Embryonic inclusion is an attractive theory of the origin of tumours promulgated by Cohnheim. It suggests that during development certain cellular elements remain in the tissues in an undeveloped state ready, under appropriate stimulation, to grow and multiply and form a tumour. These embryonic remains may have arisen from a surplus of the cells necessary to form the part in which they lie, or by inclusion of cells foreign to it but normal to adjacent structures, which have become included owing to the complexity of the developmental processes. No doubt dermoid and some other tumours are due to such a cause, and it is well ascertained that cartilaginous areas may be persistent in bones (especially in rickety subjects) and be the starting-points of chondromata (Fig. 46); moles and pigmented spots may fairly be considered as embryonic remains, and we know that these are peculiarly liable to be the seats of sarcomata. Yet it is very doubtful at present what degree of importance must be attached to Cohnheim's theory; for cancers and some other growths it is almost certainly untrue. Cohnheim himself admitted that the existence of embryonic remains cannot be demonstrated in the majority of cases; but the difficulty of such demonstration is sufficiently obvious.

FIG. 46.—Condyles and epiphysial line of a rickety femur, with a cartilage island (Bland Sutton).

Assuming the general correctness of the theory, no explanation is forthcoming as to why these embryonic cells should remain quiescent for many years and subsequently form tumours, although no doubt, whatever the exciting causes may be, they all act by bringing about increased nutrition whereby the hitherto dormant cells are awakened into activity. Perhaps injury and local irritation may become exciting causes by inducing an afflux of blood to the part. It may perhaps be pointed out here that we do not know why the beard and pubic hair should only begin to grow at puberty, since the hair-follicles are certainly present at birth.

Origin in vestiges.—Tumours may arise in connection with parts of the body which were of use during development but are functionless in the adult state, and should undergo atrophy or remain quiescent. These are known as vestiges. Others are the remains of the generative organs of the opposite sex, *e.g.* the parovarium, hydatid of Morgagni, and organ of Giraldes.

The linguo-hyal duct and processus ad testem are instances of vestiges from fœtal structures. Dermoids and cysts are commonly met with in all these situations.

Mechanical irritation and injury.—An injury, such as a blow or sprain, is sometimes credited by the patient with being the cause of a tumour, especially of the sarcomata. The effect of such an injury is very doubtful, and in some cases, at least, it probably draws the patient's attention to the part which is already the seat of a new growth, and it is hence wrongly described as the cause thereof. Long-continued irritation is undoubtedly a causative factor in the birth of some tumours, especially the epitheliomata; but the proportion of cases of all tumours traceable to mechanical injury, even as a casual relation, is so small that its etiological importance must still remain undetermined.

It is noticeable that carcinoma is especially liable to attack those parts of the body most subjected to mechanical irritation and friction. Thus, cancer of the alimentary tract usually occurs at the sides of the tongue, the narrowest part of the œsophagus, the pylorus, ilio-cæcal valve, colon, and rectum; the cervix is much more commonly affected than is the body of the uterus, and the glans penis than the sheath.

Irritated scars are especially liable to become epitheliomatous. Incontrovertible evidence of the production of epithelioma as the result of irritation seems to be furnished by the almost complete disappearance of chimney-sweep's cancer of the scrotum since

the introduction of the present mode of sweeping chimneys and the consequent diminished contact with the soot.

Parasitic origin.—It is probable that cancers and sarcomata which have many analogies with the infective granulomata, and perhaps other tumours, are dependent on parasitic infection; but at the present time experiments in inoculation and cultivation have met with uniformly negative results.

Bodies resembling Protozoa, and stated to be such by some pathologists, have been recognised as sometimes occurring in cancer cells, and also occasionally in sarcomata.

Those who deny that these "cancer bodies" are parasitic Protozoa ascribe the appearances to endogenous cell formation or to degenerative changes. Such bodies have been fully demonstrated as occurring in Paget's disease of the nipple, which is not infrequently accompanied by cancer, and they have also been found in duct cancer of the mamma. It is well known that coccidia gaining entrance to the bile ducts of the rabbit, and occasionally of man, cause cystic dilatation with papillomatous outgrowths from the wall of the ducts, closely resembling the naked-eye appearances of cancer, and microscopically similar to villous or duct cancer. The malignant nature of cancer and sarcoma and the certainty with which they induce deterioration of health and speedy death, even when not involving vital structures, are strong evidences of their parasitic origin and toxic effects.

Influence of age.—Tumours may appear at any age, but some occur with greater frequency in young or advanced life respectively. Sarcomata are more likely to occur in young patients; cancer in those past middle life; while the fibro-myoma of the uterus does not usually occur after the menopause. Innocent tumours are more usually found during the first thirty years of life than after that time, but this rule is by no means absolute.

The growth of tumours is, like that of normal tissue, directly dependent on the blood supply, which is derived both from vessels of new formation and those normal to the part in which the tumour grows.

As already stated, it is characteristic of tumour formation that the growth of the component elements is irregular, and that they are often imperfectly developed as compared with their normal prototypes. This imperfection shows itself—when growth has progressed for a time that varies in different tumours—by the often widespread degenerative changes which occur in the unstable cells.

Tumours may increase in *size* by central, general, or peripheral growth, and in the last method show a decided tendency to infiltrate the surrounding structures. Sarcomata and cancers are essentially infiltrating growths. In non-malignant tumours circumscription is the rule, and the localised mass is enclosed in a more or less dense capsule of connective tissue derived from that normal to the part which is increased as the result of irritation.

Cancers are never encapsuled, sarcomata rarely so.

The actual size attained by tumours varies in the different forms; for obvious reasons the innocent growths, which are only mechanically dangerous to life, usually attain much greater dimensions than do the malignant, and some of them, *e.g.* fibroid and fatty, may become enormous and weigh many pounds.

The size of innocent tumours is dependent in some measure upon whether the situation in which they grow offers great or little mechanical resistance to their increase.

The *rapidity* of growth varies in the different forms. Innocent tumours usually grow slowly, malignant ones quickly, although exceptions to this general law are not uncommon; and it is noticeable that the same species of tumour may grow more quickly at times than at others, although, perhaps, no cause for such a difference may be evident.

Innocent growths may attain a certain size and then remain stationary, perhaps to increase again at some future time; or, having grown slowly for a long time, they may suddenly and rapidly increase in a manner suggestive of the supervention of some malignant element. Occasionally innocent tumours undergo more or less atrophy, especially if the normal vascularity of the part is impaired (*e.g.* uterine fibroids at the menopause or after oöphorectomy), or if some mechanical cause of irritation which induces hyperæmia is removed.

As regards the rapidity of growth of malignant tumours, it may be generally stated that this is usually proportional to their vascularity and softness, and that the earlier the age at which they appear the more rapidly do they grow.

Degenerative and inflammatory changes.—Tumours are subject to all the pathological processes which may attack healthy tissues, and so common are degenerative changes that these may be regarded as normal occurrences in their life-history. As with individuals, so with cells, physiological activity and rapidity of growth (clinically evidenced in the case of tumours by their softness) entail the penalty of degeneration and death earlier than do

the reverse conditions; hence degenerative changes are more widespread and common in sarcomata and cancers than in the slowly-growing, innocent tumours.

Fatty degeneration is the most common form, but colloid, mucoid, and pigmentary also occur, and calcareous and ossific changes are often met with in some innocent growths and in sarcomata of the periosteum (Fig. 47). Hæmorrhages are common in soft vascular tumours, such as sarcomata and soft glandular cancer, the rupture of the vessels being favoured by degenerative changes in and softening of the tumour tissue, whereby the vessels are deprived of support and readily rupture under the blood-pressure or from slight injury.

Degenerative changes, softening, or hæmorrhage may result in the formation of definite cysts containing blood with broken-down, fatty, or colloid cellular elements and serous fluid.

Inflammation and suppuration of the substance of a tumour may occur, but is not common. Pedunculated growths, *e.g.* ovarian cysts, may suppurate or slough if the circulation through the pedicle be sufficiently impaired by torsion or otherwise.

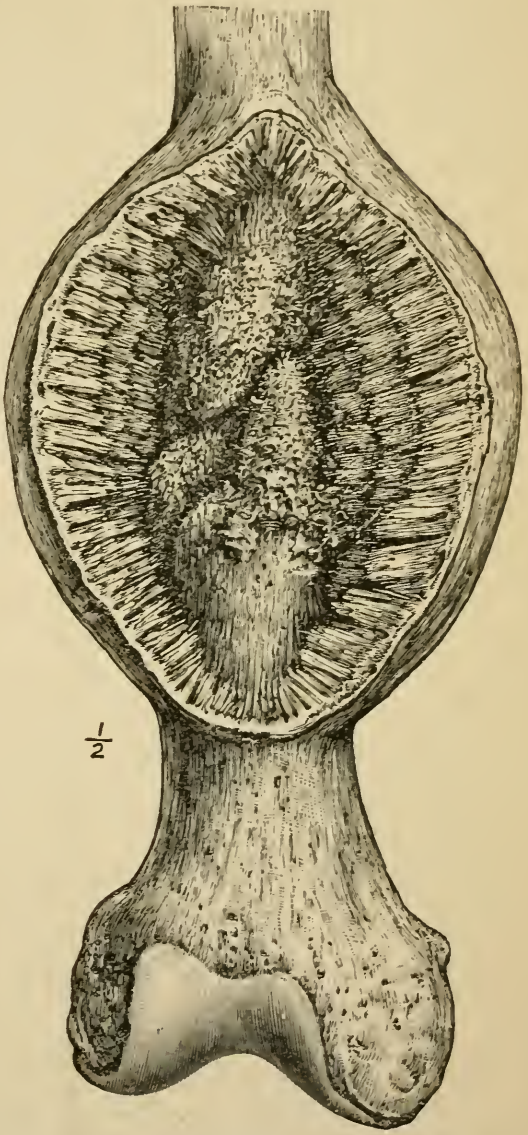

FIG. 47.—Skeleton of an ossifying periosteal sarcoma of the femur (Bland Sutton).

Subcutaneous or submucous tumours may, as the result of pressure, so impair the circulation through the parts covering them

that inflammation followed by sloughing is occasioned. The tumour substance is thus exposed and may either form a fungous mass on the surface, or may itself share in the inflammatory process, and spontaneous cure by sloughing then sometimes results if the tumour is innocent.

The number of tumours.—New growths are usually single, but they may be multiple and are sometimes very numerous, *e.g.* fatty and fibroid tumours.

In some cases more than one kind of growth is present at the same time.

The secondary manifestations of malignant growths will be subsequently dealt with.

The effects of tumours.—All neoplasms act as true parasites, deriving their sustenance from and at the expense of the host, and serving no useful purpose in return.

Innocent growths act mechanically only, while the malignant ones affect the general health as well.

Locally a tumour exerts pressure upon, and occupies the place of the tissues normal to the part, which consequently become atrophied and altered in shape; hence its effects depend on its situation and size. Even a large innocent tumour in the subcutaneous tissue will produce no ill effects when not so situated as to cause pressure on important organs (*e.g.* the trachea), whereas quite a small one implicating the central nervous system or other part of vital importance is of serious moment, not as being a tumour but mechanically, for a foreign body would be equally harmful. The more rapidly a tumour grows the more will its mechanical effects be evident, since the surrounding structures have not time to adapt themselves to the presence of the mass.

Innocent tumours never cause death unless they mechanically interfere with parts of vital importance.

The malignant growths similarly induce mechanical effects, but quite apart from these they inevitably kill. The causes of death in such cases may be quite evident, or very obscure (see p. 229).

The clinical characters of tumours.—Clinically a tumour is innocent or malignant—a point of primary importance to determine in diagnosis.

Innocent tumours are usually strictly circumscribed and are enclosed in a more or less dense capsule of areolar or fibrous tissue. They do not infiltrate the tissues among which they lie, but if subcutaneous, may lead to sloughing of the integuments through pressure. They grow slowly and may degenerate, but such

degeneration is rarely extensive or rapid. After attaining a certain size they may remain stationary indefinitely. Innocent tumours are usually freely movable, do not recur after complete removal, and never give rise to secondary deposits. Their density varies with their structure. All tumours except the sarcomata and cancers are innocent.

Malignant tumours comprise sarcomata and cancers, but the malignancy is not equal in all, nor is it shown in the same way.

A tumour may be malignant locally, generally, or both. By *local malignancy* we mean that the growth infiltrates and destroys the tissues by continuity; all forms are locally malignant, and some, *e.g.* rodent cancer, are only so. *General malignancy* is characterised by the formation of secondary deposits in the glands, internal organs or distant parts through the dissemination of the cells of the primary growth. This tendency to dissemination varies very much in the different forms of growth, thus myeloid sarcoma rarely, and melanotic sarcoma invariably, leads to secondary deposits, which may be very numerous; again, epithelioma always affects the neighbouring lymphatic glands, but deposits in the viscera are of great rarity, whereas the glandular carcinomata almost always lead to numerous visceral deposits. Sarcomata spread by the blood-stream, carcinomata by the lymphatics. In what this malignancy consists we cannot at present say; Cohnheim has suggested that the invasion of the tissues might be dependent, in the case of cancer, upon the diminished resistance offered by them in consequence of the degeneration and loss of compactness occurring with advancing years; but such an explanation is far from convincing, especially when we reflect that cancer occurring at early periods of life is remarkably malignant.

If we are prepared to admit the parasitic origin of malignant tumours, the parasites being capable of indefinite growth in the body and possessing varying degrees of virulence, we have a rational explanation of malignancy. It is quite evident that there is some inherent vice in the cells of a malignant tumour, and that the presence of the growth induces some profound alteration in general nutrition. In many cases it is quite impossible to say why a malignant tumour kills. It is true that, as in the innocent growths, death may result from implication of some part, the integrity of which is of vital importance, or may result from exhaustion consequent on sloughing or repeated and profuse hæmorrhage; but in many cases no such causes are present, and post-mortem examination reveals no lesions of a lethal nature. Take, for example, a case of epi-

thelioma of the hand with involvement of the lymphatic glands; the patient will inevitably succumb. In such a case there is certainly no interference with any essential structure, nor is there any sufficient drain on the system to cause the cachexia, emaciation, and death, and epithelial cells themselves have certainly no poisonous properties. If we accept the parasitic origin of malignant growths we may provisionally assume that, as in the case of the infective diseases, some poisonous materials, formed as the result of the activity of the parasites, are poured into the blood and produce toxic effects.

General signs of malignancy.—At first the tumour is quite localised, and some of the sarcomata may be encapsuled. As growth rapidly proceeds, the circumscription of the tumour becomes less evident as it invades and infiltrates the surrounding structures; gradual invasion and replacement of the tissues by the growth may lead to perforation of the skin, mucous membrane, or hollow viscera, and the tumour, freed of all restraint, fungates and increases still more rapidly. The size, vascularity, and softness of a malignant tumour vary with its precise nature, and, to a less extent, with its situation. Malignant tumours undergo rapid and extensive degeneration; they tend to progressively increase in size and reproduce themselves in distant parts, to recur after removal, to undermine the general health and cause death; and they never terminate in spontaneous cure.

The production of secondary deposits.—Secondary deposits always faithfully mimic in structure and behaviour the parent growth, but they are often softer and grow more rapidly. They are due to the transplantation of cells of the primary growth, which are carried to the various parts of the body by the blood-stream (Fig. 61, p. 251) or by the lymph. Cancer cells invade the lymphatic structures, whence the almost constant involvement of the glands; they also enter the blood-stream by the lymphatic ducts. Sarcomata are destitute of lymphatics, and hence dissemination occurs by the blood-stream alone, and is favoured by the anatomical characters of the vessels of these tumours (see p. 232).

Embolic transplantation explains why the secondary growths are like the primary in structure, and the frequency with which they occur in the lungs and liver.

Secondary growths may be very numerous and are often large, and each may serve as a centre for the diffusion of fresh emboli.

Classification of tumours.—In the present state of our knowledge as regards etiology, a thoroughly satisfactory classifica-

tion of tumours is impossible, but they may, for all practical purposes, be conveniently separated into groups according to their anatomical structure. It must be remembered, however, that all tumours are composed of more than one type of tissue—for instance, all have blood-vessels and connective tissue elements, and many of them are "mixed," that is, are made up of various tissues, no one of which specially predominates.

Class 1.—Tumours of the Type of Embryonic Connective Tissue

Sarcomata. (Malignant.)

Class 2.—Tumours of the Type of fully-developed Connective Tissue

Lipomata.
Fibromata.
Myxomata.
Chondromata.
Osteomata.
Odontomata.
} (Innocent.)

Class 3.—Tumours of the Type of the Higher Tissues

Myomata.
Neuromata.
Angiomata.
Lymphangiomata.
} (Innocent.)

Class 4.—Tumours of the Epithelial Type

Psammomata.
Papillomata.
Adenomata.
} (Innocent.)
Carcinomata. (Malignant.)

Class 5.—Congenital Tumours

Teratomata.
Dermoids.
} (Innocent.)

Cysts will be considered at the end of this chapter, many of them not being tumours according to the definition given (see p. 222).

Class I.—Tumours of the Type of Embryonic Connective Tissue

SARCOMATA

Distribution.—Sarcomata occur more frequently before than after thirty years of age, but no period of life is exempt. They may grow in any part of the body, but most frequently affect the skin and subcutaneous tissue, the periosteum, bones, secreting glands, and the supporting connective tissue framework of the viscera.

Morbid anatomy.—These tumours are composed of densely packed masses of nucleated cells, which vary in shape and size in the different forms to be presently mentioned, with a very delicate connective tissue stroma penetrating between the individual cells. This stroma, except in alveolar sarcoma, is always difficult of recognition and may be quite absent, in which case the cells are held together by a homogeneous intercellular substance. The softer and more rapidly-growing sarcomata are the most likely to be devoid of a stroma. As a rule sarcomata are very vascular, but there is much variation in this respect; sometimes the vessels are so large and numerous that the tumour pulsates and a bruit may be heard. Capillary vessels predominate, but numerous venous trunks—often of large size—may be present, especially in the softer growths. The vessel walls are so thin that the blood was at one time supposed merely to flow in channels between the cells without any limiting wall. This fact explains the frequency with which sarcomata grow into large veins, the occurrence of extensive hæmorrhage with the formation of blood cysts in their substance, and the production of secondary growths by embolic transplantation.

Lymphatics have never been demonstrated, and hence involvement of the lymphatic glands is by no means so constant and characteristic as in cancer; but in sarcoma of the tonsil or testis the glands are invariably involved. Sarcomata may possess, but are usually destitute of, a capsule. They tend to infiltrate the surrounding structures, growing in the direction of least resistance, and may invade and destroy the skin and fungate on the surface, forming a large, sloughy mass which may bleed profusely.

Sarcomata are specially prone to early and extensive degeneration. Fatty and myxomatous changes leading to softening and the formation of cysts are common; ossific changes are prone to occur, and are sometimes very extensive in sarcomata of bone and peri-

osteum (Fig. 47, p. 227). Cysts may also arise from hæmorrhage and degeneration with softening.

Clinical characters.—In most cases sarcomata grow rapidly, induce constitutional symptoms common to all malignant disease, become disseminated in the lungs, liver, and other parts of the body, and eventually cause death. The softness varies in the different forms; sometimes it is so marked that palpation conveys to the hand a sense of fluctuation; in other cases the density is as great as that of a fibroid tumour. The vascularity similarly differs. A sarcoma is more or less circumscribed, but may have bosses and processes extending in directions offering but little mechanical resistance to invasion. The mobility, pain, and general symptoms induced vary with the seat of the tumour. The skin over a sarcoma is often marbled by congested and dilated veins, and, as it becomes invaded by the growth, is thinned, stretched, and of a livid colour.

As the tumour grows the general health suffers, and the patient emaciates, the cachectic condition increasing as secondary growths appear.

The local and general malignancy of the sarcomata vary within wide limits. Myeloid and some forms of spindle-celled sarcoma show very little tendency to become generalised, and their local malignancy is so slight that early and free removal may be followed by permanent cure; on the other hand, while the local malignancy of melanotic sarcoma is slight, it has a greater general malignancy than has any other known tumour.

Varieties of sarcomata.—**Round-celled sarcomata** are very common and may occur at any age. The cells, which may be large or small, contain a relatively large nucleus, and are surrounded by a small quantity of homogeneous intercellular substance. In some cases there is a distinct connective-tissue stroma like that of a lymphatic gland (lympho-sarcoma or lymphoma). Round-celled sarcomata are soft and highly vascular, grow rapidly, and exhibit marked malignant properties by infiltrating the tissues and giving rise to secondary deposits. They are of a grayish colour on section and show areas of fatty degeneration, softening, and hæmorrhage. These tumours, when fresh, do not yield a milky juice on scraping.

Lympho-sarcomata are especially met with in the lymphatic glands, tonsil, testis, and retro-peritoneal tissue.

A glioma is a round-celled sarcoma with a delicate stroma like neuroglia; it chiefly affects the eye and central nervous system. A

glioma is usually soft but may be firm. It infiltrates the brain substance which appears hypertrophied, the tumour having no definite limitation. Secondary deposits are rare.

A cylindroma or plexiform sarcoma appears to be due to a degeneration of the round-celled form. The cells are arranged in columns enclosing vessels surrounded by a hyaline myxomatous tissue due to degeneration of the vessel walls and adjacent cells. This tumour is very rare and chiefly affects the brain.

Alveolar sarcoma (Fig. 48) consists of large round cells contained in the meshes of a definite fibrous stroma similar to that

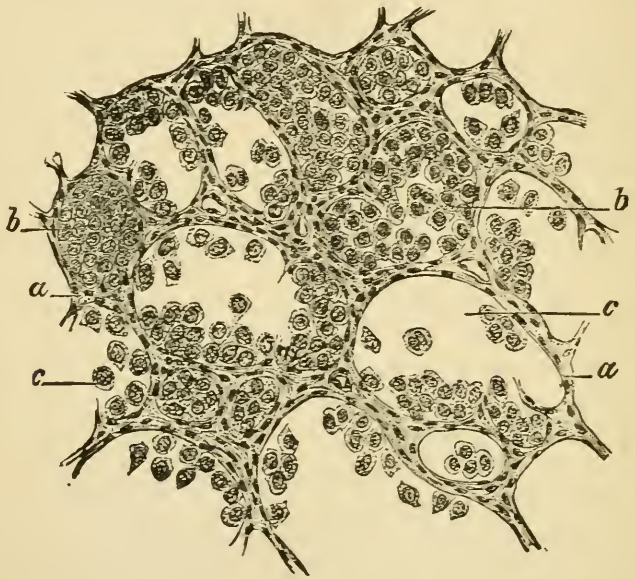

FIG. 48.—Alveolar sarcoma of a lymphatic gland (Ziegler). *a*, stroma; *b*, cell nests; *c*, alveoli with scattered cells.

seen in cancers, but differing from it in the fact that finer ramifications penetrate between the individual cells. Alveolar sarcoma is rare, and occurs in the skin, often in connection with moles. The nodules, which are rounded and hard, but do not attain a large size, are often numerous, and after a time break down, giving rise to persistent sloughy sores. Occasionally alveolar sarcoma occurs in the bones or muscles.

Spindle-celled sarcomata.—The cells, which may be small or large (Fig. 49), are oat-shaped or fusiform, with a homogeneous intercellular substance. They are arranged in more or less distinct bundles which run in all directions. Each cell has a very large nucleus, and the protoplasm is often very scanty. Rarely

the cells present cross-striations resembling voluntary muscle cells (*myosarcoma* or *rhabdomyoma*).

The stroma may be abundant and of a fibrous nature (*fibro-sarcoma*); islands of cartilage (*chondro-sarcoma*) or ossific matter (*osteo-sarcoma*) are not uncommon. Spindle-celled sarcomata are often encapsuled.

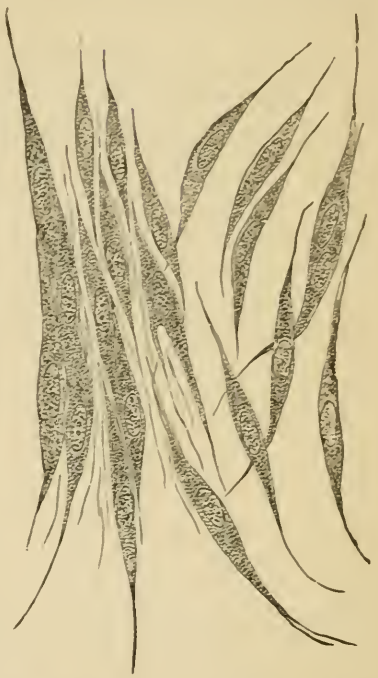

FIG. 49. — Spindle cells from a large spindle-celled sarcoma of the cheek (Ziegler).

To the naked eye a spindle-celled sarcoma resembles the round-celled, but is more dense. Clinically these tumours vary in the rapidity of their growth and malignancy. The small-celled variety usually grows slowly and closely resembles, both in naked-eye appearance and microscopically, a fibroid tumour (*recurrent fibroid*).

The softer the growth, and the more closely the cells approximate to the embryonic type, the greater the malignancy. If recurrence takes place the secondary growths progress more rapidly, show greater malignancy, and possess a lower type of cells than did the primary one. The small spindle-celled sarcoma is much less malignant than is the large-celled variety.

Melanotic sarcomata are composed chiefly of spindle cells with round cells interspersed. Dark pigment is present in and between the cells; its amount varies so that the colour of the tumour may be anything from gray to sooty-black. The surface of section may have a marbled or granite-like appearance from unequal distribution of the pigment. The tumours are often encapsuled, quite localised, and do not attain a large size; but secondary deposits may be almost universal and very numerous, especially in the liver. Melanotic sarcomata grow primarily in pigmented structures such as the choroid; they also commonly occur in abnormally pigmented spots, *e.g.* warts and moles. They are highly malignant.

Myeloid sarcoma (Fig. 50, p. 236) grows almost exclusively in the cancellous tissue of the heads of long bones, and from the alveolar borders of the jaws as one form of epulis.

The tumour is composed of round and spindle-shaped cells, with a number of multi-nucleated giant cells, which are usually branched and resemble those normally met with in the medulla of bones. The blood-vessels are numerous, and in some cases the tumour may pulsate when it has perforated the bone and infiltrated the soft structures. Cysts are not uncommon.

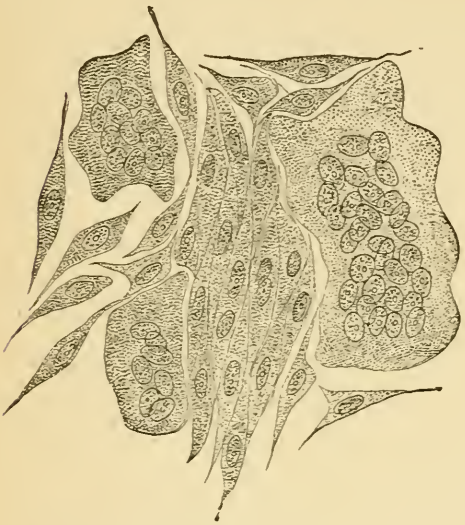

FIG. 50.—Cells from a myeloid sarcoma of the tibia (Zeigler).

On section, a myeloid sarcoma is of a pinkish hue, with maroon-coloured areas due to extravasation. Myeloid sarcomata are not very malignant locally, and secondary deposits are of great rarity.

Mixed sarcomata are very common. Gland tissue, bone, cartilage, fibrous and mucous tissue are frequently present, and sometimes, *e.g.* in the parotid and testis, give rise to the most complex growths.

Cysts of new formation or due to degeneration or hæmorrhage are frequently present.

Treatment of sarcomata.—Early and wide removal is called for in all cases where the situation or extent of the tumour holds out a chance of success. The means of removal varies with the situation Sarcomata should never be squeezed or roughly handled during an operation, for fear of detaching cells growing into the vessels, and hence inducing secondary growths by embolism.

The success of the operation depends in great measure on the variety of the sarcoma; partly also on its seat. Sarcomata of the jaw, femur, and kidney are very likely to be followed by rapid recurrence; so marked is this liability in sarcoma of the kidney in children that operative treatment appears to be useless. Gliomata of the eye are treated by enucleation of the globe; those of the brain and cord are, owing to their diffusion, not amenable to operative treatment. The treatment of carcinomata and sarcomata by Coley's fluid is referred to at p. 253.

Class 2.—Tumours of the Type of fully-developed Connective Tissue

LIPOMATA

Causes.—Fatty tumours are sometimes traceable to irritation, but in the majority of cases no cause is assignable. The tendency to their formation is sometimes hereditary, and in such cases they are often very numerous and small.

Distribution.—Commonly met with in the subcutaneous tissue about the shoulders and back, lipomata may occur in any part normally containing fat. A diffuse form, highly vascular and not encapsuled, is sometimes met with about the neck in middle-aged men, especially beer-drinkers; it may attain enormous proportions and form a complete collar. Lipomata may grow beneath mucous or serous membranes, among muscles, and in connection with the periosteum. They are not uncommon above the clavicle. A specimen in the Westminster Hospital Museum shows a large fatty tumour growing beneath the mucous membrane of the pharynx, which caused death from suffocation.

Subperitoneal lipomata may attain a very large size in the abdomen and may simulate sarcomata or ovarian cystic disease. Not infrequently small fatty tumours, originating in the subperitoneal fat, descend along the spermatic cord or into the crural canal, and simulate or conceal a hernia. In other cases fatty tumours arise in the small masses of fat which are present at the apertures of exit of the small blood-vessels in the abdomen and chest.

Parosteal lipomata are rare, and are usually, if not always, congenital; they chiefly affect the long bones and are likely to be confounded with sarcomata.

Morbid anatomy.—A fatty tumour is a lobulated mass of fat, quite smooth on the surface and contained in a delicate connective-tissue capsule, which is but slightly adherent to the tumour, though more intimately so to the overlying skin, causing it to pucker when pinched up between the finger and thumb. Fatty tumours may—apparently as the result of gravity—shift their position, or become more or less pedunculated, in which case further growth may be impeded or arrested through interference with the circulation due to torsion.

The vessels are usually small and unimportant, but may form a prominent feature of the growth (nævo-lipoma); such cases are often

congenital and widespread. Fibro-lipoma and myxo-lipoma are varieties the structure of which is indicated by their names.

Large fatty tumours may lead to ulceration of the skin through pressure. Occasionally an abscess forms in the middle of the growth.

Calcification sometimes occurs round the more central part of old lipomata, the included portion having a soapy, adipocere-like appearance.

Lipomata may attain enormous dimensions and weigh many pounds.

Clinical characters.—Fatty tumours form slowly, and are perfectly innocent; but if a small portion has been left during removal it will continue to grow. The effects produced are purely mechanical.

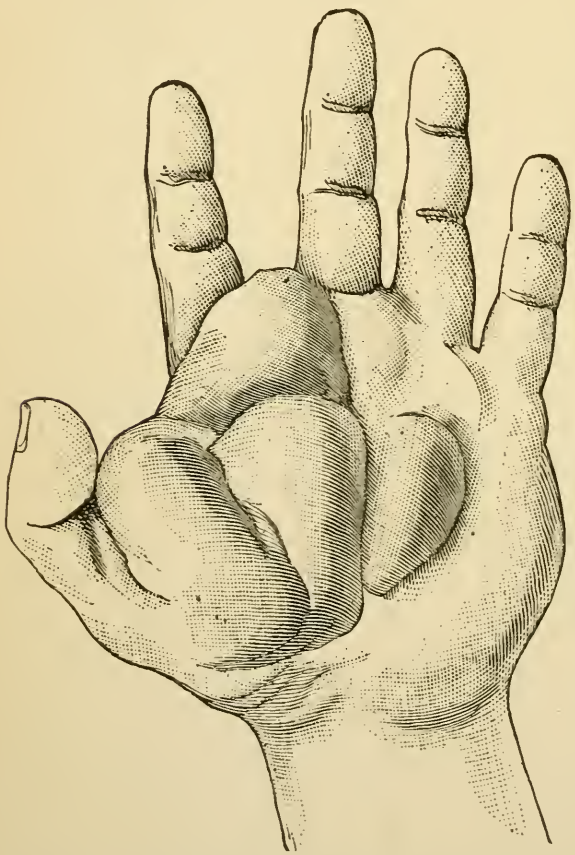

FIG. 51.—Lipoma in the palm of the hand (Bland Sutton).

A subcutaneous lipoma forms a freely movable, painless, soft, lobulated tumour, the edge of which slips away from under the finger. It is slightly adherent to the skin.

Deeply-seated lipomata cause more or less difficulty in diagnosis, which may be impossible. Fatty tumour in the palm of the hand is not uncommon, and is often congenital; it must not be mistaken for ganglion.

In the lumbo-sacral region, fatty growths sometimes conceal the sac of a spina bifida, and any operation must, in view of this contingency, be conducted with the greatest caution.

Treatment.—Diffuse lipoma and large diffuse nævo-lipomata

should not be interfered with, no treatment having hitherto proved of any avail, although liquor potassæ in the former and compression of the main vessel in the latter form have been advocated and tried. If lipomata are numerous they are usually small, remain stationary after a time, and, as a rule, need not be removed. The question of operation in the case of fatty tumours in special regions must be decided according to the circumstances of each case.

Subcutaneous lipomata are very readily and safely removed. The incision should be long enough to enable all lobules to be removed, and should open the capsule freely, when the tumour can readily be enucleated with a few touches of the knife.

FIBROMATA

Distribution.—Pure fibromata are rare tumours, but fibrous tissue enters more or less prominently into the formation of most new growths. Fibrous tumours occur in and beneath the skin as molluscum fibrosum, keloid, and painful tubercle. They may also be met with in connection with the periosteum, especially as simple epulis and naso-pharyngeal polypus, and occasionally grow in the interior of long bones. In connection with nerve-sheaths they form one variety of false neuromata. Fibroid tumours of the ovary are sometimes met with, and the common tumours of the uterus and prostate consist largely of fibrous tissue.

Morbid anatomy.—Normal fibrous tissue varies in its softness and density, and similarly fibromata are soft or hard, but they both consist of fusiform cells with large nuclei. The fibres are collected into wavy bundles, and scattered among them are numerous small round cells. These bundles of fibres are often arranged more or less concentrically. Vessels are usually small and few, but they may be very large and numerous.

Fibromata are usually encapsuled and grow slowly. They are not very prone to degenerate, but may become fatty or calcareous, and sometimes cystic from softening. They are perfectly innocent. The so-called recurrent fibroid is a spindle-celled sarcoma, and it may be extremely difficult—sometimes impossible—to say definitely from the general characters and microscopic appearance of a tumour whether it is a fibroma or a spindle-celled sarcoma.

Clinical characters.—The clinical features of fibromata depend upon their situation, and the special form of the growth.

Fibromata of bone are of the hard variety, and are clinically indistinguishable from sarcomata.

Keloid is described in chap. ii. vol. ii.

Painful subcutaneous tubercle is a small firm fibroma growing in the subcutaneous tissue, especially that of the extremities. Women are much more frequently affected than men. These tumours are usually multiple, and may be very numerous; they grow slowly, never attain a size much larger than that of a pea, and may remain stationary for many years. Very often the tubercles are so small that they do not cause any visible signs, and are only recognisable by feeling, attention being drawn to them by the pain, which is their chief characteristic. Such pain is usually paroxysmal and radiating, and may be extremely severe, suggesting the association of the growth with nervous filaments, but none such have ever been found.

Molluscum fibrosum is a soft fibroma or fibro-cellular tumour springing from the skin and subcutaneous tissue. Numerous nodules or tumours of varying size, sometimes enormous, are present in different parts of the body, and, owing to their weight, may form large pendulous masses or pedunculated growths, which are often extremely vascular. Ulceration may occur from irritation or pressure, and necessitate removal of the growth. Solitary growths of similar nature (fibro-cellular tumours) are sometimes met with, especially on the buttocks, scrotum, labia, and scalp. They are usually pedunculated, and may attain a very large size; they are very soft, and sometimes cystic. Fat often forms a prominent feature of these growths. They may be readily, and as a rule safely, removed.

Treatment.—Fibromata of bone, fibro-cellular tumours, and painful tubercles should, under ordinary circumstances, be removed.

Keloid should not be touched, as it will, although innocent, recur in the cicatrix. The tumours of molluscum fibrosum should be left alone, unless ulceration or other cause demands interference. The treatment of fibromata in other parts of the body will be discussed in the proper chapters.

MYXOMATA

Distribution.—Myxomata usually occur as pedunculated tumours—so-called polypi—in the nose, antrum, frontal sinuses, cervix uteri, and sometimes in the ear and alimentary canal. Pedunculated myxomata often grow from the labia, especially in young women.

Morbid anatomy.—Mucous tumours are composed of soft tissue made up of delicate, stellate, and branching cells, which, by

union of their processes, frequently form a beautiful network. The main bulk of the growth consists of a characteristic, glairy, mucoid material. The mucous membrane lining the cavity in which the polypus grows is reflected over its surface.

Mucous tissue often forms a prominent feature of sarcomatous growths (myxo-sarcoma), and may be met with in other tumours.

Clinical characters.—Myxomata are pinkish, soft, gelatinous, and often lobulated tumours, attached by a pedicle. They are often numerous. The symptoms they cause depend upon the situation in which they grow.

Treatment.—These tumours are easily removed by avulsion, or by the galvano-cautery loop.

FIG. 52.—Cells from a myxoma of the turbinate bones (mucous polypus of the nose), (Ziegler).

CHONDROMATA

Distribution.—Pure chondromata grow in connection with bones. The mixed sarcomatous tumours of the parotid and testis, and occasionally of other parts, frequently contain a considerable amount of embryonic cartilage. Chondromata of bone may grow in relation with the epiphyseal cartilages, in the interior of the bone, or from the perichondrium. In the large, long bones the tumour is usually situated at the end of the diaphysis. In the fingers, the centre of the shaft is more commonly affected, because in these bones cartilage islands are frequently found (Fig. 46, p. 223). The bones chiefly affected are those of the fingers, ribs, pelvis, and the lower end of the femur and upper end of the tibia. Sometimes chondromata grow in connection with the laryngeal cartilages. Chondromata, especially of the fingers, are frequently multiple (Fig. 53, p. 242),

and may attain a considerable size. They are met with in early life.

Morbid anatomy.—The tumour is encapsuled, and consists of pure hyaline cartilage, sometimes with cells of the embryonic type. It has been shown that in some cases, especially in rickety subjects, chondromata originate from included cartilage islands. These growths, sometimes lobulated, are hard and dense in structure, develop slowly, and are prone to ossification, calcification, and mucoid softening. Chondromata of a phalanx grow in the interior of the bone, which is thinned and expanded; but as the tumours increase in size the investing shell of bone may be perforated, and the tumour, no longer confined, grows more rapidly. Pure chondromata are innocent.

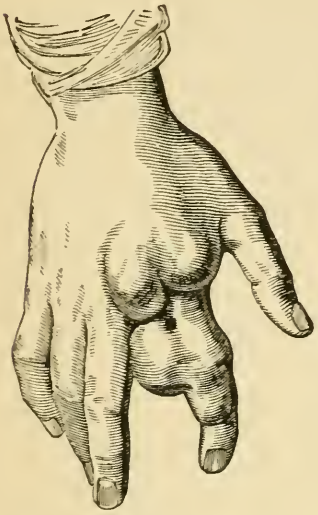

FIG. 53. — Chondromata of the second metacarpal bone and first phalanx of the index finger, which necessitated amputation (Fergusson).

The clinical characters and treatment are described in chapter v. vol. iii.

OSTEOMATA

Varieties.—Pure osteomata may be formed of compact or cancellous tissue.

Distribution.—The compact osteoma grows in connection with bones primarily developed in membrane, viz. the vertex of the skull, the frontal sinuses, clavicle, and lower jaw, being most common in the first situation. They also occur in the external auditory meatus. The cancellous osteoma is merely an ossified chondroma, and is met with in connection with the epiphysary ends of the diaphysis of long bones. A cancellous osteoma, regarded by Sutton as of inflammatory origin, is not uncommon on the dorsal aspect of the terminal phalanx of the great toe (*subungual exostosis*).

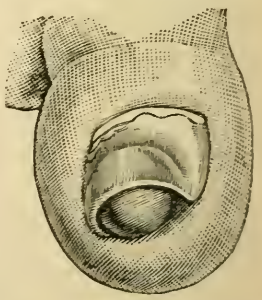

FIG. 54. — Subungual exostosis on the great toe (Bland Sutton).

Morbid anatomy.—The compact or ivory osteoma (Fig. 55) is extremely dense and hard, so that it is removed with great

difficulty. It is broadly sessile, and often resembles a limpet-shell in shape. On section the surface is ivory-like.

The cancellous or spongy osteoma (Fig. 56) is an advanced stage of growth of the ossifying chondroma. It consists of cancellous tissue similar to and continuous with that forming the interior of the bone from which it grows. The tumour, which may be rounded or nodular, is capped with cartilage undergoing ossification, and is frequently covered by a bursa. Cancellous osteomata are sessile or pedunculated; in the latter case the growth has increased peripherally, while the base has ossified and consequently ceased to enlarge. In some cases a pedunculated growth may separate by a process similar to that by which deer shed their antlers. A cancellous osteoma usually ceases to grow when the development of the bone from which it springs is completed.

FIG. 55.—Ivory osteoma of the frontal bone (Westminster Hospital Museum, No. 244. Drawn by C. H. Freeman).

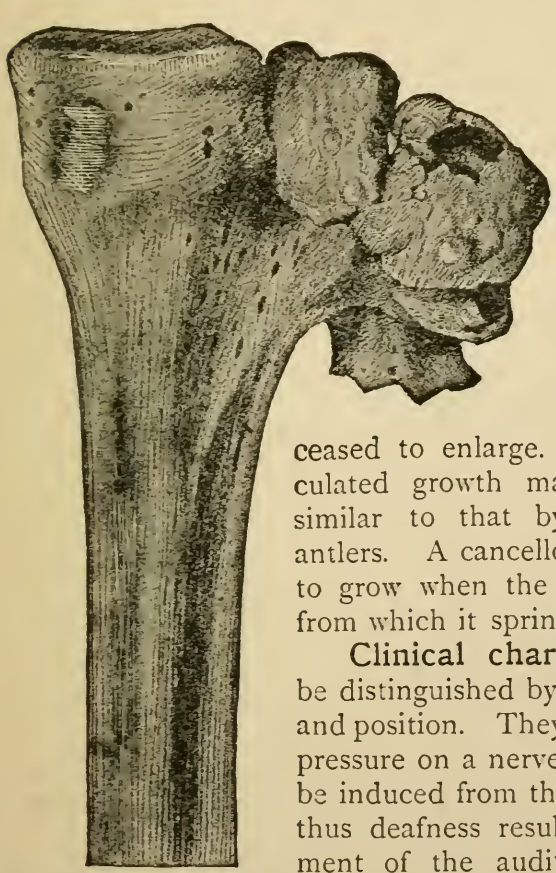

FIG. 56.—Cancellous osteoma of the upper end of the diaphysis of the tibia (Ziegler).

Clinical characters. — Osteomata may be distinguished by their density, slow growth, and position. They are painless unless causing pressure on a nerve. Special symptoms may be induced from the situation of the growth; thus deafness results in the case of involvement of the auditory meatus, and cerebral or ocular symptoms from the presence of a growth from the inner aspect of the frontal bone.

Treatment.—Removal is the only available treatment (see chap. v. vol. iii.).

ODONTOMATA

Varieties.—"An odontome is a tumour composed of dental tissues in varying proportions and different degrees of development, arising from tooth-germs, or teeth still in the process of growth."[1]

Odontomata are divided into varieties according to their origin from the different parts of the developing tooth. The epithelial odontome or enamel organ tumour, and the follicular odontome or dentigerous cyst are described with diseases of the jaws (chap. xiv. vol. iii.).

The fibrous odontome arises from thickening and overgrowth of the tooth-sac, whereby a fibrous tumour is formed, which often encloses and encapsules the developing tooth. A similar process, affecting many adjacent tooth-sacs and accompanied by ossification of the fibrous tissue which encloses numerous portions of undeveloped teeth or fragments of cement, gives rise to the **compound follicular odontome.**

Radicular odontomes consist of cement and dentine, and affect the roots of teeth after the crown has been fully developed; hence enamel is not present in this form of tumour.

Composite odontomes are composed of all the elements present in a normal tooth, but in a rudimentary state of development. They affect more than one tooth-germ and may attain a large size.

Clinical characters.—Odontomata give rise to swelling and enlargement of the jaw and all the signs of a tumour. As growth proceeds the tumour tends to come through the gum and displace the teeth in its neighbourhood.

Considerable inflammation and suppuration is excited, and hence many of these cases have been unrecognised and treated for necrosis or a tumour of a malignant nature.

Odontomes are met with in early life after the second dentition; they grow slowly, and are at first painless, but as inflammation is excited pain may be very severe. In all cases of doubt the tumour should be carefully examined and neighbouring teeth extracted before removal of a part of the jaw is decided upon.

Treatment.—The odontomes above described may usually be readily enucleated by the gouge and elevator. The operation should always be conducted through the mouth and the tumour be fully exposed by cutting down through the gum.

[1] Bland Sutton, *Tumours, Innocent and Malignant*, p. 31.

Class 3.—Tumours of the Type of the Higher Tissues

MYOMATA

Varieties and distribution.—**Striped or voluntary** muscle tissue is sometimes met with in sarcomatous growths, especially in congenital renal tumour (rhabdomyoma).

Leiomyomata or tumours of unstriped muscle are mixed with fibroid tissue and are common in the uterus. They are also met with in connection with the ovary, Fallopian tubes, broad ligament, and prostate; more rarely in the walls of the bladder, œsophagus, and other muscular organs.

Morbid anatomy.—Fibro-myomata are encapsuled tumours varying in density and rapidity of growth according to the amount of fibrous tissue as compared with the muscular. The cells are long and fusiform with elongated nuclei, and are arranged concentrically, so that the surface of section has a whorled appearance. Their vascularity varies within the widest limits. Fibro-myomata of the uterus may attain an enormous size, and are often composed of many masses bound together by areolar tissue; they may be situated within the thickness of the wall (intramural) or project beneath the peritoneal covering (subperitoneal) or mucous lining (submucous); in either of the two situations they may become pedunculated, and either cease to grow through interference with the blood-supply, or actually slough or suppurate if the latter be acutely or gradually obliterated by torsion of the pedicle.

Fibro-myomata may degenerate or soften, and may become almost completely calcified. Cysts from degeneration are common.

Clinical characters.—These tumours are quite innocent, usually grow slowly, and induce symptoms according to their situation.

Treatment.—Removal when practicable. Uterine fibro-myomata are rarely amenable to removal (see chap. xxix. vol. iii.).

NEUROMATA

A true neuroma is a tumour composed of nervous elements; a growth in connection with a nerve, but not so constructed, is a false neuroma (see Diseases of Nerves, chap. viii. vol. iii.).

ANGIOMATA

Angiomata are tumours composed of blood-vessels with a supporting framework of connective tissue. The vessels normal to

the part are dilated, their walls thickened, and they are much increased in number. Angiomata are almost always congenital, but in some situations, *e.g.* the liver, they are said to occur in later life. They may consist chiefly of capillary vessels (**capillary nævus**), of veins (**venous** or **cavernous nævus**), or of arteries (**cirsoid aneurism**). These tumours are fully described under Diseases of Blood-vessels (chap. i. vol. iii.).

LYMPHANGIOMATA

The lymphatic vessels may by dilatation and enlargement give rise to a lymphatic nævus. **Macroglossia** is practically a lymphatic nævus of the tongue (chap. xvi. vol. iii.).

Cystic dilatation of the lymph-spaces occasions a multilocular cystic tumour known as **cystic hygroma**, or may form a simple serous cyst.

These conditions are fully described under Diseases of the Lymphatics (chap. iv. vol. iii.), and Cysts (p. 260).

Class 4.—Tumours of the Epithelial Type

PSAMMOMATA

Psammoma is a rare tumour met with in connection with the choroid plexus, pituitary body, or cerebral membranes. It consists of a basis of fibrous or myxomatous tissue, with flattened epithelium cells arranged in globe-like masses and infiltrated with brain sand.

These tumours are usually quite small, perfectly innocent, and as they do not cause symptoms are only discovered post-mortem. Psammoma is classed by some pathologists either with the fibromata or sarcomata.

PAPILLOMATA

Papillomata are tumours formed on the plan of normal papillæ, and usually arise in connection with them, but some growths (*e.g.* the villous tumour of the bladder) belonging to this class occur in situations devoid of such structures.

Distribution.—Papillomata are common on the skin, the mucous surfaces of the vulva or glans penis, and about the anus, and present in different situations the characters of corns, warts, or condylomata. They are frequently traceable to dirt, moisture,

and irritation. Papillomatous growths are also met with in the urethral canal, in the bladder, in mammary and other cysts (p. 260), and in the rectum.

Morbid anatomy. — Wherever papillomata occur, their fundamental structure is the same, but they assume very different clinical appearances owing to modifications of growth and situation. A papilloma consists of a basis of connective tissue containing blood-vessels, and sometimes a lymphatic; where papillæ exist this fundamental structure is due to their enlargement, but in surfaces destitute of papillæ the connective tissue basis is an outgrowth of that normal to the submucous tissue of the part. The papillary processes are covered by layers of thickened epithelium cells which always remain limited to the surface, and do not penetrate and invade the deeper structures as in the case of cancer. Papillomata may, however, become malignant from irritation, etc., and then the epithelial invasion is present.

Papillomata of the skin are not infrequently pigmented, and sarcomatous changes may occur in them.

The epithelial covering may be very dense and hard, as in a corn, and in some cases develops into a decided horn (Fig. 57). Papillomata of mucous membranes are more usually covered with a delicate layer or layers of cells.

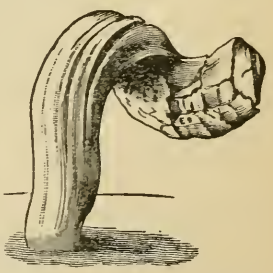

FIG. 57. — Cutaneous horn from the back of the hand (Ziegler).

The papillomatous processes may be a very insignificant part of the tumour, and only recognisable by the microscope. Thus a corn is a papilloma composed almost entirely of very dense and hard epithelium which compresses the papillæ.

The papillæ may be short, as in the ordinary papilloma of the skin; or the processes may be very long, branched, and delicate, as in villous tumours of the bladder (Fig. 58, p. 248) and duct papillomata.

The density and vascularity of papillomata vary within wide limits, as is seen when we contrast a corn with a soft and highly vascular papilloma of the bladder.

Clinical characters.—All papillomata are innocent tumours, but under certain conditions they assume malignant characters as already stated. The symptoms they induce and their physical appearance depend upon their situation, and will be described in the appropriate chapters.

Treatment.—Papillomata may be removed by excision, ligature,

cauterisation, etc. The means employed varies with the situation of the growth.

ADENOMATA

Varieties.—Adenomata are racemose or tubular, according to the variety of glands they mimic.

The racemose or acinous adenoma.—Racemose adenoma is

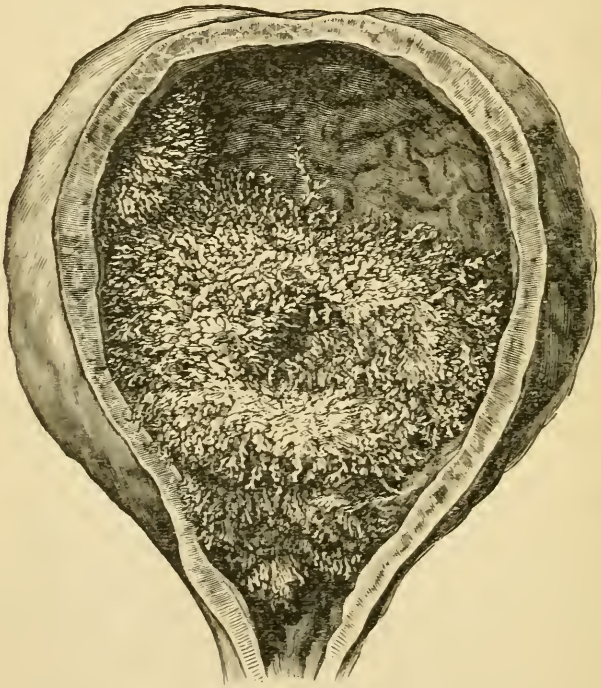

FIG. 58.—Villous tumour of the bladder (Bland Sutton).

common in the breast. Gland tissue is also met with in the mixed tumours of the parotid and testicle. Adenomata are sometimes met with in the liver, and some enlargements of the thyroid gland may be considered as adenomata. For sebaceous adenoma, see p. 263.

Morbid anatomy.—The racemose adenoma is an imperfect representation of a racemose gland, and is always perfectly distinct from the gland in which it grows; its imperfect ducts do not open on the surface, nor is the tumour capable of secreting the normal fluid. A pure adenoma, *i.e.* a tumour perfectly mimicking the normal gland, is of extreme rarity. The adenomata are structurally

composed of a stroma or basis containing slit-like, epithelium-lined spaces, and modifications in one or both of these elements give rise to tumours of different appearances and clinical effects. The slit-like spaces are lined with one or more layers of short columnar or cubical epithelium, and contain a clear serous fluid; they may be dilated into cysts, and these may contain intracystic growths. The stroma is usually composed of fibrous tissue (adeno-fibroma), or may be sarcomatous (adeno-sarcoma).

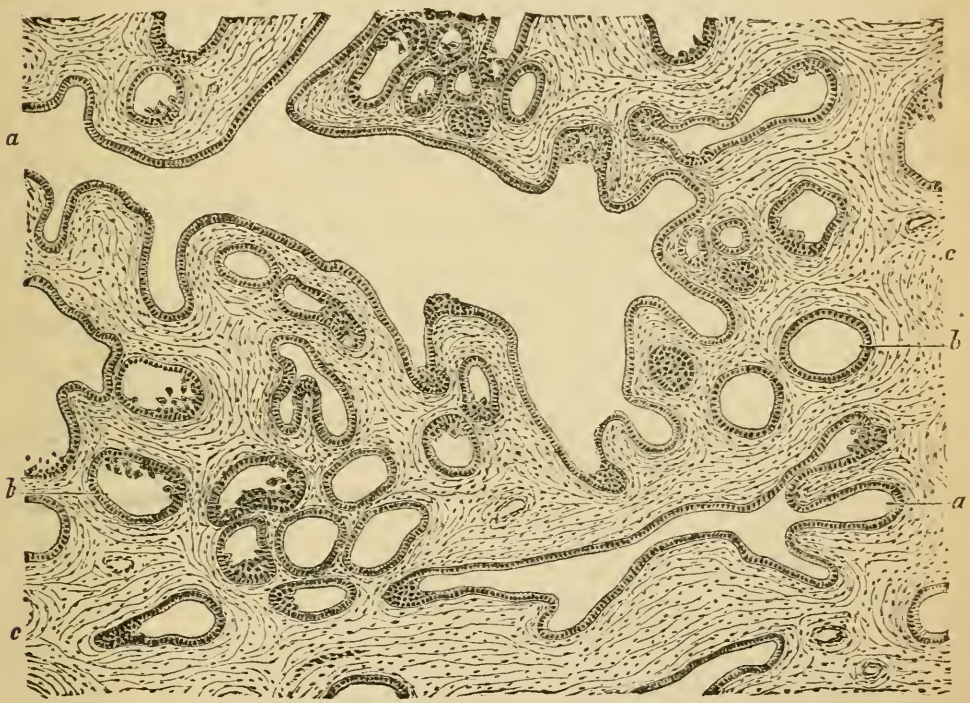

FIG. 59.—Tubular adenoma of the breast (Ziegler). *a*, longitudinal section of dilated and branching tubules; *b*, cross section of tubules; *c*, stroma.

Clinical characters.—Adenomata are firm, elastic, slowly-growing, encapsuled tumours, and may attain a very large size. If the stroma is sarcomatous the tumour will grow more quickly and assume malignant characters.

The reader is referred to the chapter on Diseases of the Breast (vol. iii.) for further information regarding these tumours.

Treatment.—Thorough removal by the knife.

Tubular adenoma.—Tubular adenomata grow from mucous membranes. The best example is the mucous polypus of the rectum. Tubular adenomata are pedunculated and consist of con-

nective tissue covered with mucous membrane, containing many columnar spaces lined by epithelium.

These growths are especially prone to become cancerous.

CARCINOMATA

A cancer is a malignant tumour originating in and composed of epithelial elements enclosed in a more or less dense alveolar meshwork of fibrous tissue. It gradually invades the adjacent structures,

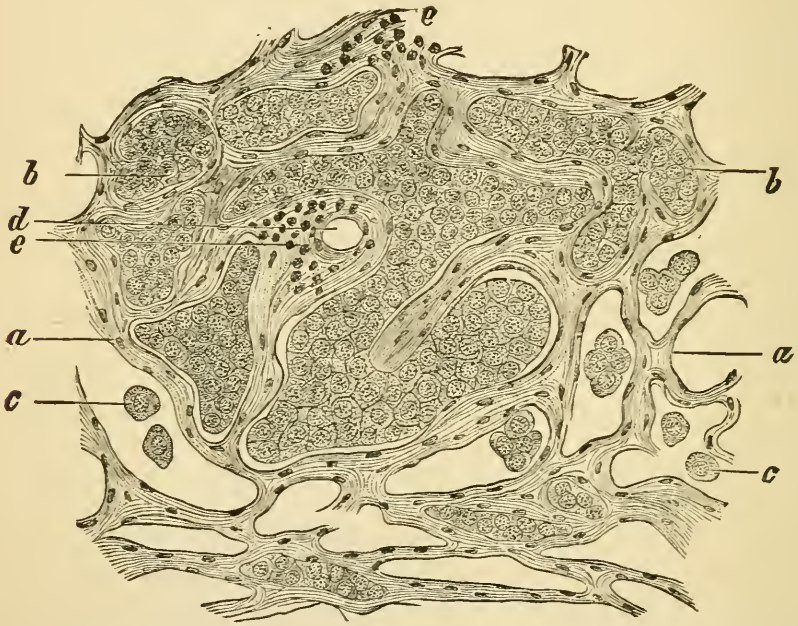

FIG. 60.—Scirrhous carcinoma of the breast (Ziegler). *a*, stroma; *b*, loculi filled with epithelium cells; *c*, isolated cancer cells; *d*, blood-vessel; *e*, cellular infiltration of the stroma.

spreading by means of the lymph-paths, and in most forms gives rise to secondary growths in the neighbouring lymphatic glands, and sometimes in the viscera and other parts of the body.

Cancer is essentially a growth of the latter half of life, but may occasionally occur in young people or even infants; it never undergoes arrest or spontaneous cure, and although its growth may be (but rarely is) extremely slow, it ends by causing death.

General structural anatomy.—The epithelium cells of a cancerous growth are atypical reproductions of the cells from which they spring, and hence differ in shape and size in the different forms. They are loosely massed together in the alveolar spaces of

the stroma, but do not, as in the adenomata, form a definite lining to the wall. It has been shown by many observers that contained in the cells, in some cases, are the "cancer-bodies," already referred to at p. 225, the precise nature and origin of which is at present doubtful. These bodies, when present, are most numerous in the region of active growth, and cannot be demonstrated in areas of degeneration, they themselves having shared in the process. All cancers are pervaded by a more or less definite and abundant fibrous stroma arranged in alveoli which communicate with one another.

In the rapidly-growing cancers the stroma is scanty, highly vascular, contains numerous small round cells, and encloses large alveolar spaces, while the hard, comparatively slowly-growing forms have an abundant and dense fibrous stroma; the epitheliomata have very little or none. The vessels and lymphatics run in the fibrous network.

Cancers do not possess any capsule, but at the advancing edge the tissues are infiltrated with round cells due to irritation; these, developing into connective tissue, form the stroma, the cancer cells spreading into it.

Invasion of the tissues by the epithelium cells is the leading characteristic of carcinomata, and is an important feature distinguishing them from adenomata or papillomata, which are perfectly innocent tumours largely composed of epithelial elements. It is supposed by some that the invasion of the tissues is favoured by the general laxity and loss of resistance occurring in advancing life; but that this has any appreciable influence is more than doubtful, for we must remember that invasion is not confined to cancers of solid organs, but occurs equally in cancer of the skin and mucous surfaces where—if resistance or its loss played an important part—invasion would be less probable than would the heaping-up of proliferated cells on the surface (see p. 229).

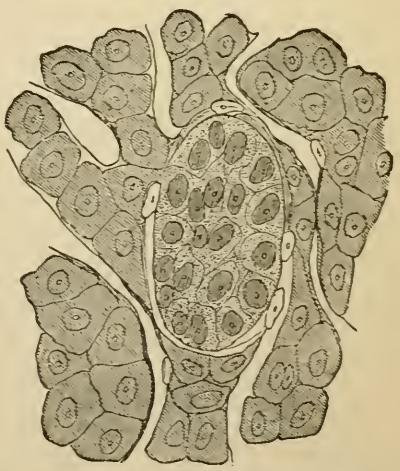

FIG. 61.—Cancerous embolus in a hepatic capillary (Ziegler).

Secondary deposits.—Cancer spreads by the lymphatics, infecting the walls of the vessels and producing secondary involvement

of the glands. Cancerous emboli may also be carried by the lymph-stream to the general circulation or may enter it directly through the vessels, and hence secondary deposits may occur in the viscera and distant parts, especially in the liver (Fig. 61, p. 251) and lungs. All forms of cancer do not show the same reproductive tendency, and in rodent cancer secondary growths are unknown.

The secondary deposits are facsimile reproductions of the primary growth, undergo the same degenerative changes, and may themselves act as centres of further infection.

Degenerative changes occur more quickly and are more widespread in cancers of rapid growth. Fatty degeneration is very common, calcification equally rare. Colloid and mucoid changes, sometimes resulting in the formation of cysts, may occur.

The soft, highly-vascular carcinomata may be the seat of hæmorrhages.

Varieties of cancer.—Normal epithelium may be divided according to its function into the glandular or secreting and the protective, and either may be the starting-point of cancer. Cancers may be thus classified:—

Glandular carcinomata—

Hard or scirrhus cancer.
Soft or encephaloid cancer.
Villous cancer.
Thyroid cancer.

Epitheliomata—

Squamous epithelioma.
Columnar epithelioma.
Rodent cancer.

Colloid cancer is the term applied to those forms which have undergone colloid degeneration; such a change is almost confined to the glandular carcinomata, and is most frequently seen in cancer of the abdominal viscera and sometimes in the breast. Colloid degeneration of epithelioma is very rare.

General treatment of cancer.—The treatment of cancer as it occurs in different parts of the body will be discussed in the appropriate chapters. In this place it will be sufficient to say that at present removal by the knife at as early a date as possible is the only means at our disposal.

Such removal must be sufficiently wide to ensure complete excision of the disease, and should the glands be involved, they must be included.

As a general rule, it may be stated that no operation should be

undertaken unless the surgeon can, so far as he is able to judge, absolutely remove the whole disease; yet in some cases, *e.g.* the tongue, it is quite justifiable and right to remove the local disease in order to ease the patient's sufferings, even when it is clear that the spread of the growth cannot be checked. If, after operation, secondary growths make their appearance, they must be treated by operation, provided their situation admits of such being undertaken. The treatment of cancer by caustics, drugs, etc. need only be mentioned to be condemned. Attempts have been made of late to produce a curative serum from cultivations of the erysipelas organism and its toxines, but so far as clinical experience goes up to the present no reliable curative results have been obtained, nor has any decided benefit been shown to accrue, although local necrosis has ensued. This treatment was suggested in view of the clinical observation that cancerous growths have been occasionally apparently cured by an attack of erysipelas. Coley's fluid is a combination of the toxines of streptococcus erysipelatis and bacillus prodigiosus. The dose of the preparation begins with half a minim and may be gradually increased. The injections are made into the substance of the tumour. Removal of the ovaries has been followed by atrophy of incurable cancer of the breast, but how this is occasioned and the value of the procedure are unknown (see chap. xxx. vol. iii.).

GLANDULAR CARCINOMATA

The difference between these two forms lies merely in the relative proportion of cells and fibrous stroma, their density depending upon the amount of the latter. The softer the tumour and the greater its vascularity, the more rapid is its growth and the more evident are its malignant tendencies. All gradations may be met with between a dense and hard scirrhus and the softest and most rapidly growing encephaloid. For clinical purposes it will be convenient to describe the characters of the hard and soft varieties separately.

Hard glandular cancer or scirrhus is chiefly met with in the breast, but may also occur in the pancreas, prostate, skin, or at the pylorus. It usually occurs after the age of forty, sometimes earlier. In most cases the growth is comparatively slow and degeneration of the cells occurs early, probably on account of the relative poorness of the blood supply. Sometimes this form of cancer of the breast may take many years to grow, or remain apparently quiescent (*atrophic scirrhus*). At first the tumour is

more or less circumscribed, of stony density but freely movable; later on it becomes adherent to the deep and superficial structures and may infiltrate the skin and fungate on the surface, or give rise to a deep, unhealthy, indurated ulcer (Fig. 62) covered by fatty granulations, or showing a tendency to slough. As the skin is approached the contraction of the fibrous elements of the growth causes dimpling. The lymphatic glands are involved and secondary tumours of softer nature and more rapid growth appear in distant organs.

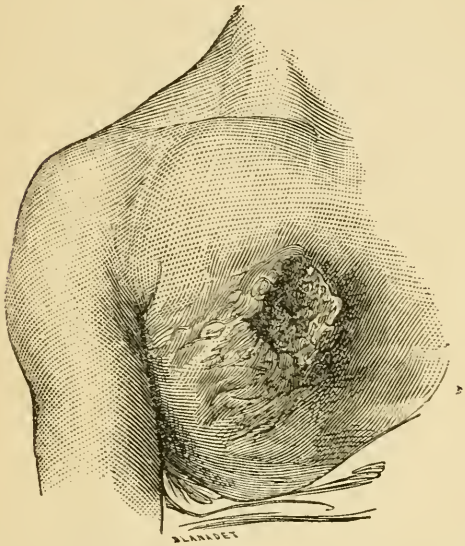

FIG. 62.—Ulcerating scirrhus of the breast, with invasion of the surrounding parts (Follin).

On section, a typical scirrhus has the appearance of an unripe pear or turnip; it is very dense, grayish-white in colour, and flecked with streaks and spots of yellow due to fatty degeneration. Radiating from the margin are fine processes and grayish-white lines indicating the direction in which the growth is spreading. The surface of a section does not bulge but is rather cupped, and when scraped yields a milky juice which is rich in epithelial cells.

Soft glandular cancer or encephaloid is rare, and chiefly affects the breast, testes, or liver. The component cells may be very large; the fibrous stroma is scanty, highly vascular, and encloses large alveolar spaces. Growth is very rapid and as the skin is invaded the tumour fungates on the surface, giving rise to a large sloughy mass which may bleed profusely. Clinically and macroscopically these growths closely resemble the soft sarcomata, the microscope being necessary for diagnosis. They are highly malignant.

On section the cut surface bulges, and the tumour is of a pink colour and brain-like appearance. It is very soft and sometimes pulpy; large hæmorrhages may be present and these may have broken down the tumour tissue into a diffluent mass.

Duct or villous cancer is a rare form met with in the breast. It is characterised by the distension of the ducts into cysts containing papillary growths, the epithelium of which proliferates and invading the walls of the ducts grows into the breast substance. The tumour

clinically runs much the same course as other glandular cancers, and is moderately soft. It is much less malignant than the other forms, and the glands are involved late.

Thyroid cancer is a very rare form met with in the thyroid gland, mimicking that organ in its structure and reproducing similar tumours as secondary deposits (see chap. xiii. vol. iii.).

THE EPITHELIOMATA

Squamous epithelioma is often traceable to some long

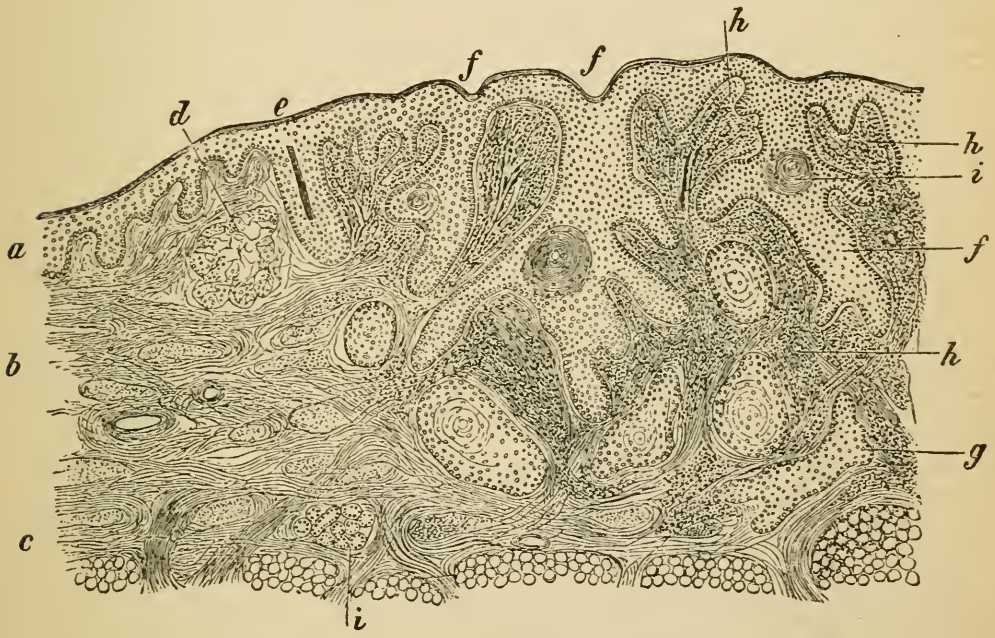

FIG. 63.—Section from an epithelioma of the skin (×20), (Ziegler). *a*, epidermis; *b*, corium; *c*, subcutaneous areolar tissue; *d*, sebaceous gland; *e*, hair follicle; *f*, cancerous ingrowths of the epidermis; *g*, deep-set cancerous cell groups; *h*, proliferating fibrous tissue; *i* (above), cancer cell nest or epidermic globe; *i* (below), sweat gland.

continued irritation and usually occurs at parts where mechanical irritation is most frequent. It is common at the sides of the tongue, at the muco-cutaneous margin of the lips (usually the lower), in scars, and about the vulva, glans penis, etc.

Morbid anatomy.—The cells are large and flattened with a definite nucleus, or sometimes more than one owing to endogenous growth. They grow into the lymph spaces as solid anastomosing cylinders from which they may sometimes be squeezed out like the secretion in a comedo. As growth proceeds the cells pass

along the lymphatics and affect the glands, but secondary deposits elsewhere are of the greatest rarity. Through mutual compression the cells are flattened, and at the seats of most rapid growth "bird's-nest" collections are formed. This arrangement, which may also be seen in papillomata, is due to rapid central proliferation, the peripheral cells being compressed while the central ones are often fatty; hence the appearance of these nests is somewhat that of an onion in section (Fig. 63, *i*, p. 255). The stroma is never a marked feature of the growth; it is formed by the connective elements of the tissues among which the epithelium cells penetrate. Owing to irritation the tissues are infiltrated with numerous, small, round cells. Growth towards the surface gives rise to a circumscribed, fungous mass raised above the level of the surrounding parts and distinctly indurated. In such cases the cells may rapidly break down and an ulcer result, or they may lose water by evaporation and become dense and horny like the surface of a wart (cornified or warty epithelioma).

Clinical characters.—Epithelioma may begin as an ulcer, a persistent fissure, or a small tubercle which soon ulcerates. There is always marked induration and the glands are quickly involved. Pain is never very severe and often absent. An epitheliomatous ulcer, when quite small, may be covered with a scab, but as it increases in size this is no longer possible, and the floor, which is sloughy and may be covered with fatty, unhealthy granulations, becomes exposed; the edges are raised, everted, sometimes undermined, and always indurated. As ulceration extends, the soft structures are destroyed and vessels may be opened; the dense structures are usually respected. Ulceration on the surface may keep pace with growth in the deeper parts so that no definite tumour is ever formed, the disease assuming the characters of a malignant ulcer rather than of tumour growth.

Columnar epithelioma is not a common growth, but may originate in the columnar epithelium of the stomach, intestines, rectum, or uterus, and is sometimes found growing from the lining membrane of the antrum.

Morbid anatomy.—On microscopic examination these growths resemble the tubular adenomata, being composed of columnar cells arranged in columns and enclosing gland-like spaces, or completely filling the lumen of the tubular stroma. The cells, unlike those of adenomata, spread beyond the tubular structures and infiltrate the surrounding tissues. Secondary deposits are common in this form of epithelioma.

Clinical characters.—Columnar cancer, when growing in the gut, tends to completely encircle it and to spread to the surrounding parts. It is clearly demarcated to the naked eye, and has a raised, usually rounded and dense margin, but is ulcerated in the centre. The mass grows more slowly than squamous cancer, but will prove equally fatal.

Rodent cancer or rodent ulcer.—Rodent ulcer is an epitheliomatous cancer of slight malignancy often remaining stationary for many years. It is characterised by local persistence, but never becomes disseminated or directly affects the general health. It is very rare before the thirtieth, and most usually begins after the fiftieth year.

Distribution.—Rodent cancer always begins in the skin; its origin is attributed by different observers to the hair-follicles, sweat-ducts, or sebaceous glands, and it may probably originate in any of these structures. Any part of the skin may be affected but rodent ulcer is far more frequently seen on the lower eyelid or side of the nose than in all other parts put together.

Morbid anatomy.—Microscopically a rodent cancer is found to consist of columns of small cells of an epithelial character containing oval nuclei embedded in an imperfect connective-tissue stroma. The cells never form "nests" as in squamous cancer, and are very delicate, so that their outline is sometimes difficult to distinguish. They may be vacuolated, but do not form a fungous or warty mass, such as is seen in squamous cancer.

Clinical characters.—Rodent ulcer begins as a pimple, or flattened, brownish tubercle, which ulcerates. When quite small the ulcer is covered with a hard, dry scab, removal of which exposes a deep, punched-out, crater-like, florid surface. The scab quickly re-forms. The ulcer may remain in this condition for years, or may gradually increase in area until the most extensive damage is inflicted. The surface of the ulcer is grayish-red in colour, smooth, glazed, destitute of granulations, and secretes a very scanty, thin, watery discharge. The centre is depressed, the margin slightly raised but free from induration.

The tissues are gradually destroyed irrespective of their nature or density, and in bad cases the bones at the base of the skull may be destroyed and the brain exposed. It is characteristic of rodent ulcer that it spares nothing, spreading, however, in superficial extent rather than in depth. The glands are not involved, and secondary deposits do not occur. The course is painless and unattended by constitutional cachexia, the patient usually dying of some other

disease. In some cases epithelium may grow from the margin of the skin and cause partial healing, but this is only temporary and is never sound or accompanied by cicatrisation.

Treatment.—Complete and free removal of the ulcer is the only treatment, and, if efficiently performed, is followed by a permanent cure. If the disease be too extensive for this nothing can be done beyond keeping the surface clean, or attempting to arrest the progress of the ulceration by the application of caustic pastes or the strong mineral acids.

Class 5.—Congenital Tumours

TERATOMATA

A teratoma is a congenital tumour composed of all kinds of tissue in a more or less imperfect state of development and frequently mixed together in a confused mass. Some of these tumours are instances of fœtal inclusion, a parasitic and imperfectly developed fœtus being grafted on, or included in the body of the living and developed twin; in other cases the teratoma is due to imperfect differentiation of the tissues of a single fœtus. Teratomata may attain an enormous size and are met with about the head or neck, or internally, usually in connection with the generative organs. The most common form is the congenital sacral tumour (see p. 316).

DERMOID TUMOURS

A dermoid is a tumour of congenital origin containing skin or mucous membrane and their appendages or modifications. These tumours are usually met with in the young, but from their position or size may not attract attention for some years.

Varieties and distribution.—**Sequestration dermoids** arise from the inclusion among the mature tissues of detached portions of epiblast at the points of coalescence of the skin during embryonic life. Sacral dermoids, scrotal dermoids, and those along the middle line of the trunk arise at the line of union of the two lateral halves of the body. During the development of the face epiblastic rudiments may be shut off at the naso-orbital fissure, or along the line of junction of the maxillary plates, or that of the superior plate with the fronto-nasal process (see Development of the Face, p 278). Dermoids are most common at the outer angle of the eye but sometimes occur at the inner angle or in the naso-

facial sulcus. They are also met with in the palate, at the root of the nose, and in connection with the ear. At an early period of development the scalp and dura mater are in contact, but are eventually separated by the growth of the bony walls between them; if, during this process, a portion of the skin remains included, a dermoid tumour attached to the dura mater by a fibrous pedicle and projecting through a hole in the vertex will result, owing to the incomplete development (Fig. 64).

Tubulo-dermoids is the name given by Bland Sutton to dermoid tumours arising in connection with obsolete canals and clefts. Thus, dermoids of the neck may arise from epiblastic inclusion in the line of

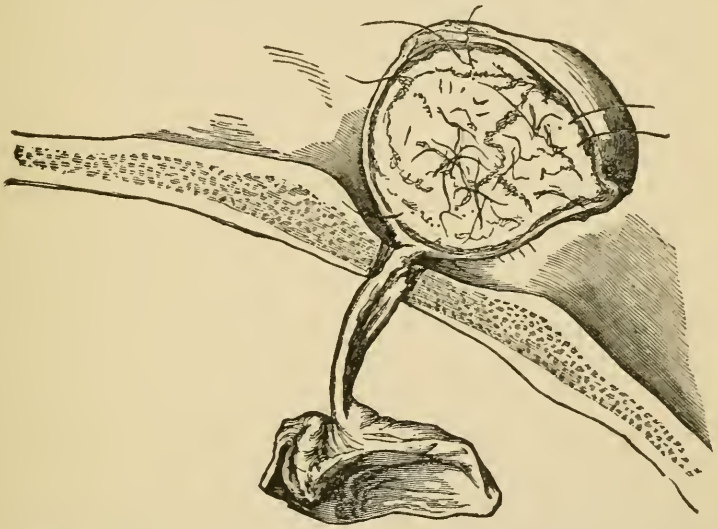

FIG. 64.—Dermoid of the scalp connected by a pedicle with the dura mater (Bland Sutton, from a specimen in the museum of the Middlesex Hospital).

the branchial clefts. Lingual dermoids arise in connection with the linguo-hyal duct; and occasionally a dermoid is met with behind the rectum in association with the post-anal duct, which may also be the origin of the congenital sacral tumour.

Ovarian dermoids (see chap. xxix. vol. iii.).

Morbid anatomy.—The morbid anatomy of dermoids varies somewhat with the situation in which they occur, and will be referred to in the appropriate sections.

They all consist of a fibrous wall which is more or less completely lined with imperfect skin or mucous membrane covered by several layers of epithelium cells. Sebaceous and sweat-glands are both present and the cyst is filled with sebaceous material and shed epithelium scales, cholesterine, and hair. The amount of hair

varies: ovarian dermoids contain the most and longest, sequestration dermoids usually only a few short, fine hairs. When the tumours are old the hair may fall or turn gray. The sebaceous glands are often much larger and more numerous than in normal skin. Teeth are common in ovarian dermoids but are not met with in those due to sequestration of epiblastic elements. Sutton has described a dermoid in which he found an imperfect mamma.

Clinical characters.—Dermoid tumours are quite innocent, appear at an early age, and grow slowly. A dermoid usually forms a more or less rounded, elastic tumour resembling a sebaceous cyst. The situation of the tumour is usually an important element in the diagnosis.

Treatment.—Dermoids should be removed unless their situation precludes the justifiability of operative interference.

Cysts

Definition.—A cyst is a sac of fibrous tissue containing fluid or soft pultaceous matter. The term is a clinical one but is not pathological, embracing as it does many conditions of very varying nature and mode of formation, some of which are tumours in the proper sense of the term while others are not.

General morbid anatomy.—The wall of any cyst is composed of fibrous tissue elements which may be of new formation, or may consist of the fibrous wall of the duct or acinus to dilatation of which the formation of the cyst is due. In any case the wall is strengthened by fibrous tissue of new formation as the result of irritation, and hence it may become much increased in thickness. The walls of some cysts, however, are extremely thin. If a cyst has arisen from dilatation of a tube containing muscular tissue, traces of it may be found in the wall but the greater part will have undergone atrophy. Externally the wall is connected to the surrounding parts by delicate areolar tissue so that the cyst may be easily enucleated; but in some cases—especially if inflammation has been excited—the wall is very adherent. Cysts arising in connection with a space lined with epithelium or endothelium are themselves similarly lined, unless the epithelium has undergone atrophy and destruction from pressure; otherwise, the interior is destitute of cellular lining. The wall of a cyst may enclose a single loculus, or the interior may be traversed by bands or septa dividing it up into numerous loculi which communicate with each other. A cyst is said to be *compound* when it consists of numerous loculi which do not communicate, *e.g.*

some ovarian tumours and cystic hygroma. In such cases some of the loculi may eventually communicate by giving way of the wall dividing them. Papillomatous and solid outgrowths of the wall may project into the cavity of the cyst, as in some cases of ovarian and mammary tumour (*Proliferous cyst*).

The contents of a cyst vary with its origin; but, whatever fluid it may originally contain, this tends with time to become more dense and inspissated and to lose its original character. The contents may be mixed with inflammatory products, should the wall have been the seat of such a change.

The size of cysts varies very much; the largest are met with in the ovary. Sometimes growth is arrested by calcification of the cyst wall or some other cause according to the nature of the cyst. The wall may inflame and suppuration ensue.

General clinical characters.—Cysts are perfectly innocent. They usually grow slowly in the direction of least resistance and thus may send long processes among the muscles, etc. A cyst forms a rounded, smooth, tense, elastic tumour, which, when superficial, may be translucent if the contents are clear and the walls thin. There is no pain unless a nerve be pressed upon, and the symptoms are due to mechanical pressure only. If deep-seated or very tense, or with thick calcareous walls, it may be impossible to diagnose a cyst from a solid tumour without puncture. A cyst may remain stationary for years, and may gradually get somewhat smaller as the fluid contents inspissate. In other cases it may undergo spontaneous cure by rupture or suppuration, or may cause serious and fatal results from its position or size.

General treatment.—The treatment applicable to a cyst in any given region will be indicated in the proper place; it is here sufficient to point out the various methods of treatment which are applicable under suitable conditions.

Simple aspiration is a temporary means only, although in a few instances it may lead to permanent cure. Aspiration and injection with iodine, carbolic, etc., is useful in some forms, especially hydrocele of the tunica vaginalis. Subcutaneous rupture is sometimes successful in curing ganglion, and acupuncture is useful in hydrocele in infants. Ranulæ and mucous cysts are best treated by removal of a portion of the wall and destruction of the epithelial lining by caustic.

Excision, when possible, is the best method. If other means fail, and excision cannot be undertaken, the cyst must be laid open and allowed to heal by granulation.

Origin and varieties.—Cysts are classified according to their methods of origin.

(*a*) **Retention cysts.**—If a duct which opens on the surface of the skin or mucous membrane becomes obstructed the secretion will accumulate behind it, and a cyst result. The obstruction is usually only partial, and may be continuous or intermittent. Such cysts are limited by the distended duct or acinus wall and are lined by the epithelium common to the part. The wall is increased in thickness by new fibrous tissue. The contents are the normal secretion, which in old cases is inspissated and altered in appearance, and may be calcareous or fatty, and perhaps mixed with inflammatory products.

Retention cysts may arise in connection with the ducts, acini or tubules of the breast, pancreas and salivary glands, testicle, kidney, liver, and sebaceous or Meibomian glands.

Retention cysts of mucous glands are chiefly met with beneath the mucous membrane of the mouth and lips, and in the canal of the cervix uteri, more rarely in the stomach and intestines.

The reader is referred to the special chapters for their diagnosis and treatment.

Sebaceous cysts (syn. *atheromatous cysts or wens*).—A sebaceous cyst is a tumour formed by the accumulation of sebum within a fibrous capsule of varying thickness, formed by the distended gland wall and new fibrous tissue. Sometimes the duct is obliterated, at others it is patent; and no doubt some sebaceous cysts arise in connection with sebaceous glandular elements in the skin, which, from imperfect development, have never been provided with an excretory duct. Sebaceous cysts usually contain a cheesy material which may undergo decomposition, soften, and become extremely offensive; in other cases it gradually escapes through the minute opening on the skin, and dries upon the surface. By continued escape a definite sebaceous horn may result and attain a considerable size (Fig. 65). If the cyst wall inflames, it becomes adherent to the surrounding structures, the skin becomes thinned and ultimately gives way, and the purulent and cheesy contents are evacuated. In such an event spontaneous cure may result, or the discharge continue for a long time; should the cavity become septic, a spreading destructive inflammation may result, and a foul surface makes its appearance, which suggests malignant ulceration. Malignant disease does occasionally attack the epithelial lining of sebaceous cysts. Sebaceous cysts may be single or numerous, and are commonly met with about the scalp. They are, unless inflamed, quite painless, of slow

growth, rounded in outline, and movable on subjacent parts but attached to the skin. A small black depression or pimple may indicate the position of the duct, and this may be covered over with a small black mass of extruded and dried sebum.

Sometimes a complex tumour is met with composed mainly of overgrown sebaceous glands, some of which may be dilated by sebum (sebaceous adenoma). Such a tumour may inflame and ulcerate and form a fungating cancer-like mass. So-called lipoma nasi consists largely of sebaceous glands (see chap. x. vol. iii.).

Treatment.—Sebaceous cysts are readily removed by enucleation

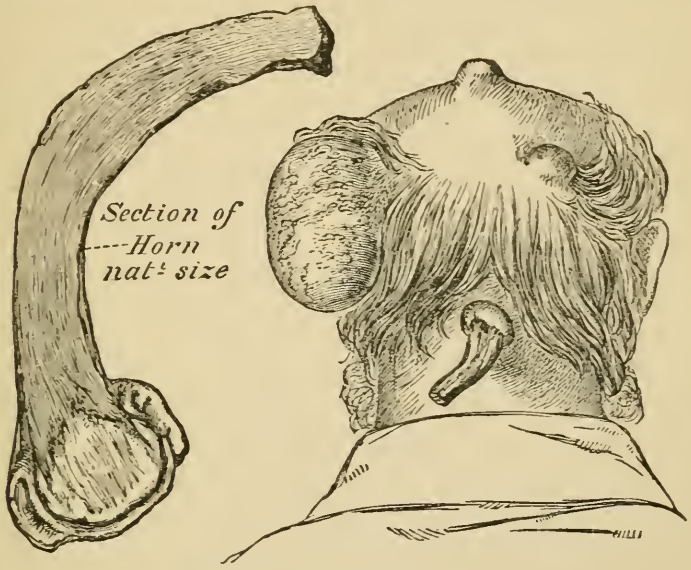

FIG. 65.—Sebaceous tumours in scalp and horn (Bryant).

after an incision has been made over them. If inflamed, and the skin over the most prominent part is thinned and much adherent to the tumour this may be removed with it. Sometimes an inflamed cyst cannot be completely removed and it is then necessary to lay it freely open, evacuate the contents, and sharp-spoon the interior of the cyst. If any part of the secreting surface of the wall be left reaccumulation of the sebaceous matter is to be expected.

(*b*) **Exudation cysts.**—Accumulation of fluid in closed spaces having no excretory duct, in lymphatic spaces, or in functionless canals, gives rise to an exudation cyst. The structure is similar to that of a retention cyst but the lining of the wall varies with the origin of the cyst. The contents are clear serum.

Dilatation of the lymph-spaces results in the formation of a

simple serous cyst or false bursa. Cystic hygroma is formed in this way. Bursal cysts, ganglion, hydrocele, and Morrant Baker's cysts (arising, as diverticula, from synovial membranes) are examples of exudation cysts into closed serous cavities. Thyroid cysts, and those of the ovary, parovarium, and paroöphoron, some forms of encysted hydrocele, cysts of the neck, and the rare cysts of the urachus and vitello-intestinal duct, belong to this group, in which meningoceles are also included by some writers.

(*c*) **Extravasation cysts.**—Hæmorrhage into the tissues or into the substance of a tumour or closed sac, may result in the formation of a tumour bounded by a dense fibrous wall and containing altered blood-clot (hæmatoma, hæmatocele).

(*d*) **Implantation cysts.**—It occasionally happens that, as the result of injury, a portion of the superficial tissues is driven into and remains embedded in the deeper structures. Should this continue to grow fluid may be exuded into it, and thus a cyst is formed. Occasionally, instead of a cyst, a solid tumour having the histological characters of the embedded tissue, results.

(*e*) **Parasitic cysts.**—Cysts due to the presence of the tænia echinococcus or hydatid are common, and are described in chap. viii. vol. iii.

Cysticercus cellulosæ, the larval form of tænia solium, is occasionally met with in man, and is contained in a small thin-walled cyst. It chiefly affects the muscles (see chap. viii. vol. iii.).

CHAPTER XII

Deformities

Deformities of the Spinal Column

Spina bifida

Definition.—A spina bifida is a congenital gap in the spinal column through which some of the contents of the canal bulge and give rise to a tumour.

Etiology.—The primary cause of spina bifida like that of other congenital deformities is unknown. Whatever the cause be, it acts in most cases at an early period of fœtal life and prevents a proper differentiation of the epiblastic layers from which the cord and cutaneous structures are developed, so that these remain in contact, and the vertebral laminæ are undeveloped. Spina bifida is often associated with hydrocephalus or talipes, and in the cervical region with crania bifida or anencephalous.

Morbid anatomy.—Spina bifida is most common in the lumbo-sacral region of the column, rarest in the dorsal region, and in the cervical is often associated with crania bifida. In extremely rare instances the cleft has been through the bodies, the tumour projecting anteriorly. Occasionally only one vertebra is affected, but usually several neural arches remain undeveloped; the gap thus varies in size—a matter of importance in reference to treatment.

The sac or tumour is centrally situated and is round or oval in shape, with its long axis parallel with that of the column. It is usually sessile with perhaps slight constriction at the base, but in some cases (chiefly those of simple meningocele with a small cleft) it is pedunculated. Usually small at birth, the tumour may rapidly increase and attain a large size unless it bursts. It often presents

at or near its most prominent part a depression, median furrow, or umbilicus, indicating the point at which the cord and nerves blend with the sac-wall, which in consequence yields less readily to the pressure of the cerebro-spinal fluid (Fig. 66). The interior of the sac may be loculated, and its contour show furrows and lobulations indicative of such a condition; these loculi usually communicate with one another by small rounded openings.

Coverings.—Normal skin very rarely covers the entire tumour; it is usually only present at the base and ends abruptly or gradually in a more or less membranous tissue which completes the covering. At the summit this membranous area may have a raw appearance, and is very liable to rupture or slough. In those cases in which skin forms a complete covering it is usually smooth, shiny, and scar-like at the summit of the sac, where it may slough. The dura mater is blended with the superficial covering, and the arachnoid passes into the base of the neck of the sac, and, in nearly all cases, is then lost.

Contents.—The sac may or may not contain nervous tissue, according to the variety of spina bifida. The fluid is ordinary cerebro-spinal fluid, clear, colourless, of low specific gravity, usually about 1007; it is faintly alkaline, and contains a trace of albumen, a copper-reducing substance, and about 1 per cent of solid matter consisting chiefly of sodium chloride.

Varieties.—Simple spinal meningocele.—About 12 per cent of all cases belong to this form in which the tumour contains fluid only, the cord and nerves lying within the canal. The cleft is usually small and in some cases there may be none, the protrusion then occurring between the adjacent laminæ. The tumour is not infrequently pedunculated, and is often covered with normal skin. Spontaneous cure may result, and treatment is more successful and less fraught with danger than in any other form. This form is alone suited to treatment by excision.

Meningo-myelocele occurs in about 86 per cent of all cases. The cord or nerves of the cauda equina enter the sac, and blending with its wall may be almost indistinguishable from it. The nerves cross the sac in order to reach their foramina of exit from the spinal canal. Where the cord blends with the sac a depression is usually present, as mentioned above.

Syringo-myelocele makes up the remaining 2 per cent of spina bifida. This very rare form is due to dilatation of the central canal of the cord. The posterior part of the cord and its nerves lie in the sac, the latter being intimately blended with it.

Diagnosis.—The general signs, appearances, and situation of the tumour have been already described. The margins of the cleft and the continuity of the tumour with the spinal canal may be made out by examination of its base. Fluctuation is present, and the tenseness of the tumour, which is partly reducible, varies with circumstances. If the child is held upright the tension is increased, but is diminished if the pelvis is raised; tension is also increased during strong expiration, as in crying, but is diminished in forced inspiration. In some cases an impulse may be obtained at the anterior fontanelle when pressure is exerted on the tumour.

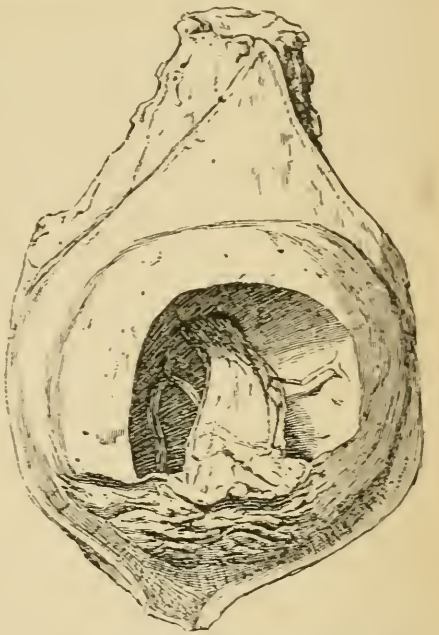

FIG. 66.—Myelo-meningocele in the lumbo-sacral region of a young child. The nerves of the cauda equina form a flat band attached to the lower part of the sac which is puckered; some of the nerves pass to the sac wall at the sides. (Westminster Hospital Museum, No. 1279. Drawn by C. H. Freeman.)

Course and prognosis.—The prognosis of spina bifida is bad; most cases gradually increase in size, and eventually rupture. Spontaneous cure occasionally results, especially in pedunculated spinal meningocele; in such cases the small opening in the canal closes, the cyst is cut off, and constitutes one form of false spina bifida. Death may occur from convulsions, rupture and sloughing of the sac, meningitis, or marasmus.

The prognosis is naturally influenced by the degree and situation of the deformity, the state of the coverings of the sac, and the general health of the child. In rare cases adult life is reached.

Treatment.—Unless the circumstances of the case demand more immediate treatment, nothing of a curative nature should be undertaken until the child is two months old, the tumour being merely protected and supported by the adjustment of pads of lint or Gamgee tissue.

Simple aspiration of the sac is not advisable, as sudden death not infrequently results. Ligature and excision of the sac should never be undertaken since they are both dangerous procedures, and under any circumstances can only be practised in cases of simple

meningocele, which it is usually impossible to distinguish from meningo-myelocele.

The only radical treatment to be recommended is by the injection of Morton's fluid (iodine, grs. 10; iodide of potassium, grs. 30; glycerine, ℥i.), and this is not applicable to all cases. If the sac-wall is unhealthy and very thin, if the general health is bad, and if convulsions, paralysis, or marked hydrocephalus is present, treatment by Morton's fluid is dangerous. The most favourable cases are those in which the sac is small and its coverings sound, the general health good, and the condition unaccompanied by complications. In properly selected cases the injection of Morton's fluid is successful in about 50 per cent. The injection is made as follows:—

A fine and perfectly clean needle is passed into the sac; it is very important that the puncture should be made at the base through healthy skin. If the sac is very tense a little of the fluid may be drawn off, but otherwise this is unnecessary. From half to one and a half drachms of the fluid is then slowly injected, the needle is withdrawn, and the puncture closed with collodion. The tumour should then be covered with a wool dressing carefully bandaged on to afford support. The fluid diffuses slowly, and remains limited to the sac if the patient be kept quiet in the recumbent position. Following the injection there may be some slight signs of inflammation, but these soon disappear, and in successful cases the sac gradually contracts, hardens and is eventually obliterated by new fibrous tissue, as the result of chronic inflammation. The nerves traversing the sac are incorporated in this new tissue but are not usually injuriously compressed, although such an unfortunate result may occur with consequent paralysis and anæsthesia. The injection may require repeating, but this should not be done for ten days or a fortnight after the first operation. The dangers of the proceeding are shock, sloughing and rupture of the sac, convulsions and meningitis, and the friends should be informed of these possible results and of the probable chances of success before the operation is undertaken.

In cases unsuitable for radical treatment protection of and support to the tumour is all that can be done. If very thin the sac may be advantageously covered with a thin layer of gauze and collodion.

SPINA BIFIDA OCCULTA

Spina bifida occulta is the name given to a rare condition in which there is a congenital deficiency of the neural arches in the

lumbar or sacral region, but without any hernial projection of the contents of the spinal canal. The gap is filled up with membrane and is indicated by a slight depression on the surface, or at least by the absence of the normal number of spinous processes. Growing in and round the depression is a considerable amount of hair, which is usually dark (the skin being also pigmented), and sometimes many inches in length. Similar growths of hair have been also noted in the cervical region, and sometimes the trunk is generally more hairy than natural. In conjunction with spina bifida occulta observers have recorded the occurrence of talipes, perforating ulcer, and caries of the metatarsal bones, associated with overgrowth of the muscular coats of the arteries, thrombosis of the veins, and degenerative changes in the nerves. Virchow considers that the growth of hair at the seat of the deformity is the result of local irritation due to disturbance during the development of the spinal column; and it may be assumed, from the frequency of the association, that the other conditions mentioned have a central origin.

SPINAL CURVATURE

Causes.—Curvatures of the spine, unconnected with caries of the bones forming it, may occur under the following conditions:—

(1) In consequence of faulty posture during the years of active growth, by which the spine is habitually placed in, and encouraged to assume, an abnormal position. This is especially operative in those growing rapidly and of feeble muscular development.

(2) As a natural consequence of old age and the following of any occupation which entails continued stooping (agricultural labourers), the carrying of heavy weights upon the shoulders, or some sedentary calling in which the patient bends over a desk. Such causes are especially causative of kyphosis.

(3) As compensation for, or as the more direct result of, some pathological condition such as hip-disease or empyema.

(4) As the result of rickets.

Varieties.—The following forms of curvature are met with:—

Antero-posterior curvatures.—(*a*) lordosis, in which the convexity of the curve is forwards; (*b*) kyphosis, in which the convexity of the curve is backwards.

Lateral curvature or scoliosis.

The angular curvature of Pott's disease, and that which may occur in cases of tumour of the column are considered under those conditions.

LORDOSIS

In lordosis or "saddle-back" the convexity of the curve looks forwards.

Etiology.—This deformity is met with in the lumbar region and is, like kyphosis, an exaggeration of the normal curve.

In children capable of walking rickets may lead to an increase of the lumbar curve; but in the great majority of cases lordosis is compensatory, due either to the deformity of hip-disease, of congenital or unreduced dislocation of the femur, or of rickety or other deformity of the pelvis or lower limbs.

Angular curvature in any part of the column leads to the formation of secondary lordotic curves, the situation of which will depend upon the seat of the angular deformity.

Temporary slight lordosis may be due to increase of the abdominal contents (*e.g.* pregnancy, ascites), necessitating additional curvature of the lumbar spine in order that the upright position may be maintained—such cases do not come under the care of the surgeon; transitory lordosis may also be due to muscular irritation of reflex origin.

Anatomy.—In recent and temporary lordosis no permanent changes are met with, but in old standing cases the spines of the vertebræ are approximated, the discs are compressed behind but thickened in front, and the muscles and ligaments are contracted and shortened on the concave side, but are relaxed and stretched on the convex. In rheumatic patients the ligaments may be ossified, and osteophytes may be present on the bodies and processes of the vertebræ.

In cases of lordosis the patient walks very erect with the shoulders thrown well back and the belly protruded.

Treatment.—Treatment must be directed to an avoidance of those causes which may, if neglected, eventually lead to the deformity or to a rectification of them as far as may be when lordosis is actually present. Rickety children must be kept in the recumbent position in a wicker cradle, until by general treatment the pathological conditions have been remedied and the spine is able to bear the necessary weight.

KYPHOSIS

In kyphosis the convexity of the curvature is directed backwards. It is usually limited to the dorsal region and is an

exaggeration of the normal posterior curvature in this situation, but it may affect the whole column.

Causes.—The normal curvatures of the spinal column are but slightly marked until a child begins to assume the upright position. Before this time kyphosis may be induced if the child is nursed in the upright position, since the column is not strong enough to support the weight of the trunk. Rickets tends to cause kyphosis, because the column is unstable and there is muscular and general weakness. Prolonged stooping positions, especially if combined with insufficient bodily exercise may (especially in girls at the age of puberty when they are rapidly developing) cause bending of the column; but in such cases scoliosis is much more common than kyphosis. In advanced life most people show some degree of kyphosis.

The subjects of chronic bronchitis often become very round-shouldered, and rheumatoid arthritis may lead to a like result.

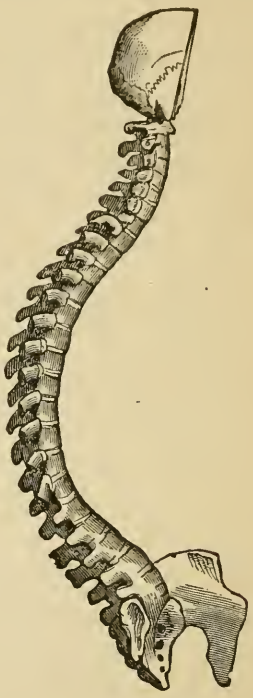

FIG. 67.—Kyphosis affecting the entire spinal column (Follin).

Anatomy.—In early cases, which are capable of complete cure by appropriate means, no changes are met with, but in permanent and old standing cases there will be separation of the bones and lengthening with relaxation of the ligaments on the convex and the reverse conditions on the concave side of the bend. The separation of the bodies behind and approximation in front is compensated for by growth or atrophy of the intervening discs; but if the kyphosis is present during the stage of growth of the spine, the bodies themselves become wedge-shaped, the apex of the wedge being anterior. Changes such as these necessarily render the condition permanent. In the subjects of rheumatism or rheumatoid arthritis the ligaments may be ossified. In consequence of the changes in the spine, the thorax is increased in its antero-posterior, and diminished in its lateral diameters; the sternum may be slightly bent with the convexity backwards. The head is depressed, the chin approaches the sternum, and the shoulders are much rounded and raised, so that the lower angles of the scapulæ project. In marked cases of kyphosis the

patient looks upon the ground, being unable to look straight forwards.

Treatment.—In cases occurring in young subjects the treatment is practically that described under scoliosis; the kyphosis of old age requires none. Weak or rickety children should rest in the recumbent position, and the muscular tone and development should be encouraged by shampooing, douching, friction, and, if the child be old enough and when the muscular power has been increased by these means, by mild gymnastics.

SCOLIOSIS

Etiology.—Scoliosis or lateral curvature of the spine usually occurs in rapidly growing girls about the age of puberty, and is, in some cases, traceable to an inherited tendency. The rapid growth at this age is often out of proportion to the strength of the muscles and supporting structures of the column, and the curvature is further favoured by anæmia and the more sedentary habits of the patient with the onset of the menses and sexual development. Slouching and incorrect attitudes, especially if maintained for long periods during piano-playing, drawing, or reading, are factors of importance in the production of the deformity, since the base of support of the column (the pelvis) is movable, and if it is tilted to one side there is a compensatory curvature of the column which may become permanent.

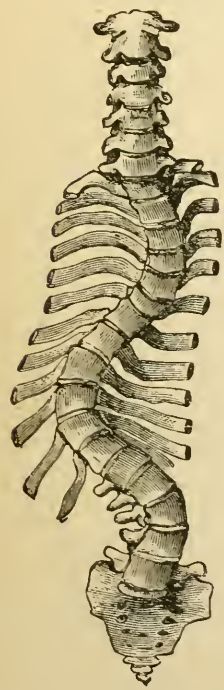

Fig. 68.—Scoliosis (Follin).

Rickets may induce scoliosis unless great care be taken to take weight off the column.

Empyema may, if the chest fall in, be compensated for by lateral curvature.

Inequality in length of the lower limbs is an important cause, and its early rectification will arrest the deformity; the same applies to errors of refraction, especially when the focus of the two eyes differs.

Anatomy and clinical characters.—Scoliosis is a spiral twist of the vertebral column, the vertebræ rotating round a vertical axis. The degree of associated lateral deviation varies. The rotation of the vertebræ is greatest at the centre of the

curve, and here may amount to as much as a quarter of a circle, gradually becoming less above and below. The rotation takes place towards the convexity of the curve, the spines of the vertebræ turning to the concavity, so that the line of these does not indicate the degree of curvature.

The initial curve is in the lumbar region with the convexity to the left, the compensatory dorsal curvature being to the right; in some cases the curvatures are in the opposite direction. As the muscles are unable to support the column in the straight position, considerable strain is thrown upon the ligaments, which become stretched on the convex side and shortened on the concave. The bodies of the vertebræ and the discs on the concave side tend to atrophy from pressure, whereas on the convex side they grow more rapidly; and hence become wedge-shaped with the base at the convex side; the result of this will be to render the curvature permanent. The actual nature and extent of these anatomical changes naturally depend upon the degree and duration of the scoliosis, and upon the means taken to remedy the defect; they may be so slight as to leave no permanent changes.

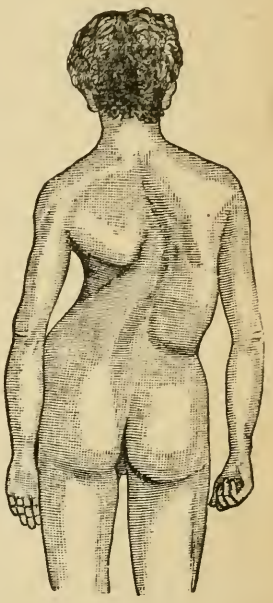

FIG. 69.—Scoliosis (Tillmans).

The altered direction of the vertebræ necessarily causes a corresponding alteration in the shape of the chest and in the patient's configuration.

The child is probably brought to the surgeon because her shoulder or hip is "growing out," the former on the convex side, and the latter on the concave side of the dorsal curve (Fig. 69).

The right shoulder is raised and the scapula rendered prominent, since the ribs are carried backwards. The right ribs are bent at their angles and the intercostal spaces are widened, while on the left the angles are opened out. Viewed from the front, the chest will be found to be flattened on the convex side, but bulged and the breast prominent on the concave side (Fig. 70, p. 274).

On the convex side the clavicle is considerably curved; the sternum is oblique and its lower end is prominent; the pelvis is not usually altered unless the patient has been the subject of rickets.

In the early stages of the deformity the spine can be com-

pletely straightened out by extension, but later on, when the beforementioned changes have occurred in the bones, discs, and ligaments, no complete rectification of the deformity can occur. Scoliotic patients are usually pale, feeble, and easily tired; they are liable to bronchial affections, which may prove serious, owing to the undeveloped state of the chest.

Prognosis.—The prognosis in scoliosis depends upon the circumstances of each individual case. If the condition is marked, and there is evident axial and lateral deviation, this will remain permanently; but, speaking generally, it may be said that if examination shows that the spine can be straightened by extension,

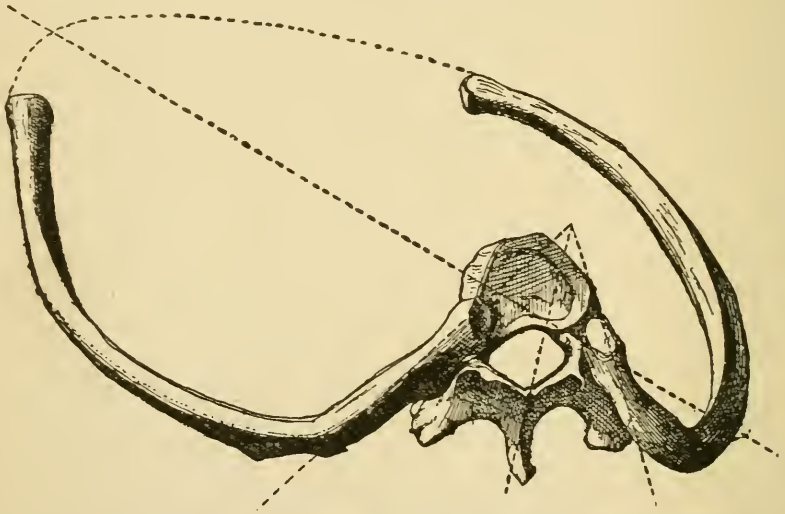

FIG. 70.—Illustrating the alteration in shape of the ribs, and the deviation of the transverse diameter of the thorax in scoliosis (Tubby, after Rédard)

the prognosis is good and the patient may grow up without any very evident deformity. The later the age at which the scoliosis appears the better. If the curvature is dependent on inequality of the lower limbs, it will soon disappear if this be rectified by a high-soled boot for the shorter limb.

Treatment.—As the great majority of cases of scoliosis are dependent upon weakness of the supporting structures of the spine, improvement in the nutrition and strength of these is of the first importance. The application of spinal supports is likely to do more harm than good, since it aims at straightening the column by mechanical means instead of inducing the muscles and ligaments to efficiently perform their work. Artificial support may, however, be occasionally employed with advantage if the patient complains of much pain, or

if, in spite of other treatment, the deformity is rapidly increasing; a proplastic jacket is to be preferred, since it can be readily removed.

Faulty habits and slouching attitudes must be at once corrected. Moderate gymnastics, friction, and massage to the spinal muscles, especially those on the convex side of the curve, with cold douching, must be daily employed, and the patient should take outdoor exercise but never to fatigue. If the curvature is advancing and the patient is weak, anæmic, and growing rapidly, it will be necessary for her to assume the recumbent position for some hours daily, and the gymnastic exercises must be limited in amount to her powers.

In all cases the general health must be encouraged by fresh air, good food, and tonics.

Deformities of the Head and Neck

Crania Bifida

Owing to a deficiency in the cranium its contents may protrude through the opening, and give rise to a definite tumour. If only the meninges project the sac is filled with cerebro-spinal fluid, and may be quite translucent (*meningocele*); but in many cases more or less cerebral substance is present (*encephalocele*), making the tumour more dense and solid than in the case of simple meningocele; it will pulsate with respiration. The skin over such a tumour may be quite normal, but it is sometimes nævoid; it often ulcerates, and eventually yields, so that the child dies of meningitis.

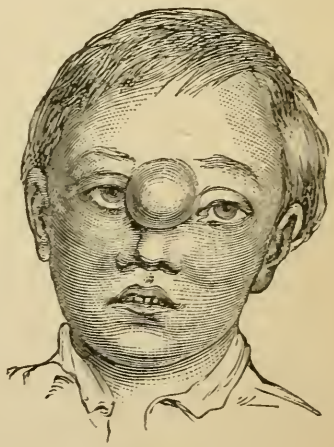

Fig. 70A.—Meningocele at the root of the nose (Bryant, from a patient of Mr. Poland).

Crania bifida is usually met with in the occipital region midway between the foramen magnum and the posterior fontanelle in the middle line; it also occurs at the root of the nose (Fig. 70A), at either fontanelle, or through the base of the skull into the nose or pharynx.

Spina bifida, hydrocephalus, and other deformities may be associated.

Diagnosis.—The congenital origin of the tumour and its position will lead the surgeon to suspect its nature. The fluid may be partly displaced into the cranial cavity by steady pressure, and a small meningocele may be almost completely reducible. If brain matter is

present, it may be in sufficient quantity to render the tumour solid, and make respiratory pulsation very evident, but in some cases the brain matter is very small in amount, and cannot be certainly detected.

A small meningocele is by no means incompatible with life, although it infinitely rarely undergoes spontaneous cure. Encephaloceles tend to steadily increase in size. Most children with crania bifida die within a few weeks of birth.

Treatment.—This consists in supporting the tumour and preventing it from being injured. If the case is undoubtedly one of simple meningocele, and the child is otherwise healthy, the sac may be excised, the base of it being very carefully sutured across the opening; the presence of any brain matter negatives such an operation.

Injection with Morton's fluid should not be undertaken.

TORTICOLLIS OR WRYNECK

Torticollis is false or true, and the latter may be spasmodic or permanent.

False torticollis is the condition in which the head is voluntarily bent to one side in consequence of some local disease. It is seen in cases of spinal caries, enlarged glands, cellulitis, and abscess about the neck, or as simple stiff neck from rheumatism or cold. In these cases the muscles are contracted on the side of the disease leading to the wryneck, and if such disease be removed the contraction is at once relaxed. The treatment must have reference to the cause.

True torticollis.—Spasmodic torticollis is usually unilateral, and may affect the sterno-mastoid muscle only, or also the posterior deep cervical muscles, in which case the head is drawn backwards and laterally (*Retrocollis*). The spasm may be *clonic* or *tonic*, and often intermits, so that the patient may be free from it for weeks together, or, although persistent, it may be very much worse sometimes than at others, and often is so if the patient's attention is drawn to it. Unless the case is very bad there is no spasm during sleep. The spasm does not cause fatigue of the muscles, although cramp and pain are experienced after a time. The muscles do not undergo any pathological change.

Spasmodic torticollis is usually seen in young women, especially those who are highly neurotic (hysterical); it is sometimes dependent upon reflex irritation, such as decayed teeth, enlarged glands, and the like, and such a cause should always be sought for and removed. Irritation may also directly affect the spinal accessory nerve or its roots.

Treatment.—Anti-spasmodics, combined with tonics, should be

given a fair trial. Conium, morphia, cannabis indica, and the bromides should be tried, combined with the use of galvanism. If this treatment fails, the spinal accessory nerve should be divided, and if after this the posterior muscles give trouble, the posterior cervical nerves may be similarly dealt with.

Permanent torticollis.—Contraction of one sterno-mastoid, maintaining the head in a fixed position may be due to injury of the muscle at birth, which causes a hæmatoma and subsequent contraction of the muscle (*sterno-mastoid tumour*, see p. 140, vol. ii.), but this association is by no means constant, and although the association as cause and effect is maintained by some writers, it is denied by others. Syphilitic myositis and pronounced astigmatism are also causative agents. In some cases no cause can be assigned.

The sterno-mastoid is the muscle primarily at fault, and may be the only one affected, but in some cases the deep muscles are also implicated. The cervical fascia is often contracted. The muscle is dense, hard, and stands out prominently, the two heads of origin being well defined. The occiput is drawn to the same, and the face to the opposite side; the chin and shoulder are raised, and the clavicular curves are specially pronounced.

FIG. 71.—Congenital torticollis, showing asymmetry of the face in a child three years ten months old (Tubby).

This continued contraction of the muscles of one side of the neck may produce a lateral deviation of the cervical spine, with a compensatory dorsal curve. In marked congenital cases the face on the affected side may not be fully developed, as can be shown by careful measurement (Fig. 71).

Treatment.—Any contributory cause, such as syphilis or astigmatism, must be corrected, and this, coupled with daily manipulation and massage, and the use of a leather collar or support, may correct the deformity in early cases.

If tenotomy is needful, this should be performed by the open method, since the subcutaneous is, in view of the important relations of the sterno-mastoid, too dangerous a procedure. An incision is made about 1½ inch above the clavicle, and the tendons of the sterno-mastoid are fully exposed, and divided by short snips with the scissors. Care must be taken that the anterior and external

jugular veins and the vessels beneath the muscle are not damaged. The wound should be closed, and the head may be at once immobilised in the correct position on a suitable apparatus. In old cases, where the cervical fascia is contracted, this may be carefully divided, but if the contraction is but slight, it will usually be overcome in time without operation. The apparatus should be worn continuously for about three weeks, and during the day for another week. At the end of a fortnight gentle massage should be employed to the side of the neck.

Complete cure of the deformity can hardly be looked for if a lateral curvature of the cervical spine has been induced.

Congenital Malformations of the Mouth, Lips, and Face

Normal development.—The face is developed by the partial union of a vertical plate known as the fronto-nasal process, with two superior and two inferior lateral plates—the maxillary and mandibular. The fronto-nasal process grows downwards from the base of the skull between the ocular vesicles, and at its lower end is deeply notched on each side to form the nostril; the lateral margins of the notches form the alæ nasi while the central portion which separates the two nostrils gives rise to the tip of the nose, the columella, the central grooved portion of the upper lip, and the intermaxillary bone with the incisor teeth.

The maxillary plates grow forwards towards the middle, where they unite with the fronto-nasal process on each side, and thus form the upper lip and cheek above the level of the angle of the mouth. The mandibular and maxillary plates unite posteriorly to the angle of the mouth; in front of this point the former give rise to the lower lip. Imperfect union of any of these processes will result in hare-lip, cleft lower lip, macrostoma, or cleft cheek, according to the position at which such imperfection occurs.

The palate is formed by the union of the intermaxillary bone with the maxillary plates, and behind by that of the palatine processes of the maxillary plates, to which is joined above the fronto-nasal process forming the septum nasi. The union of the component structures of the palate and upper lip takes place from before backwards.

Hare-lip

Hare-lip is due to failure of union of the lower end of the fronto-nasal process with that portion of the maxillary plate which forms

the lateral part of the upper lip. The deformity may be single or double; the cleft may be partial, or extend right into the nostril, and may or may not be associated with cleft palate. Hare-lip is more common in boys than girls and is not infrequently hereditary. The term hare-lip is incorrect, as the cleft in the hare's lip is central.

Single hare-lip occurs more usually on the left side. The general appearances of the single and double deformities are well shown in the figures. If the cleft be single, and especially if it be slight, there is practically no difficulty in feeding the child, but in the more severe cases and those associated with cleft palate such

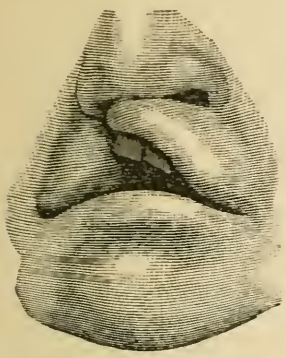

FIG. 72.—Single hare-lip on the right side with a cleft of the alveolar margin. The intermaxillary bone forms a marked prominence to the left of the cleft. (Follin.)

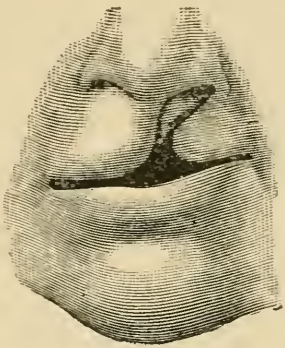

FIG. 73.—Single hare-lip on the left side extending into the nostril and through the alveolar border. (Follin.)

FIG. 74.—Double hare-lip with complete cleft palate. The intermaxillary bone covered with skin is attached to the tip of the nose. The alæ nasi are broadened and the nose flattened. (Drawn by C. H. Freeman.)

difficulty is great, and unless care be taken the child will suffer severely from malnutrition. The method of feeding is the same as in cases of cleft palate (see p. 282).

Operation for single hare-lip.—Clefts of the lip should be operated on when the child is about a month or six weeks old, and should always be closed before the seventh month, *i.e.* before primary dentition begins. The operation may, if the parents so desire, be safely performed within a few days of birth, but the risk of failure is greater and slight cases only should be undertaken at this age. The operation consists in paring and uniting the edges of the cleft with silkworm gut and horsehair. The precise operation and lines of incision must vary with the circumstances of the case. The accompanying figures (Figs. 75 to 79, p. 280) show the methods which are most usually applicable. In all cases the lip

must be freely detached from the subjacent bone by snipping through the mucous membrane and soft structures, so that there may be no undue tension on the sutures. Hæmorrhage is prevented by grasping

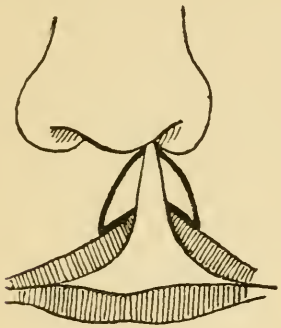

FIG. 75.—Rose's operation for single hare-lip. The dark lines indicate the lines of incision.

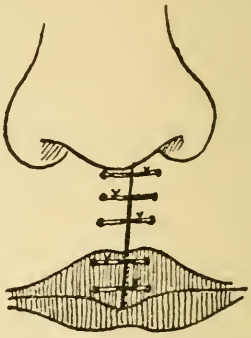

FIG. 76.—Rose's operation completed by suturing of the freshened surfaces.

the lip between the finger and thumb, and when the edges of the cleft have been pared, the sutures, which are passed almost through the thickness of the lip but not through the mucous membrane, will effectually control the vessels, so that no ligatures are either necessary or advisable. Accurate suturing is essential, so that the normal contour of the lip should be attained. When the cleft has been united the lip should be everted and the mucous membrane drawn

FIG. 77.—Mirault's operation for single hare-lip. The dark line indicates the line of incision.

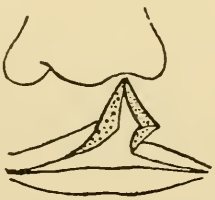

FIG. 78.—Mirault's operation, showing the freshened surfaces.

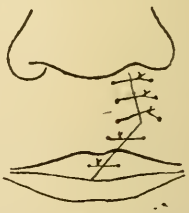

FIG. 79.—Mirault's operation concluded by suturing of the freshened surfaces.

together by a few horse-hair sutures. When the operation is completed the parts are covered with a thin layer of salicylic wool and collodion, which may remain untouched for four or five days; the sutures should be removed at the end of a week. If the gap is wide and there is the least tension on the sutures, the centre of a narrow bandage is placed on the forehead, and the ends are carried beneath the occiput and then brought forwards over the lip, one end is split, and the two pieces are drawn through slits in the

opposite end; by drawing on the two ends the edges of the cleft are brought together, and the bandage is then knotted behind the occiput.

For the success of the operation it is essential that the child should be in good health.

Operation for double hare-lip.—In cases of double hare-lip the premaxillary bone covered by the skin, which should form the columella of the nose and the central portion of the upper lip, is attached to the end of the nose and helps to fill up the gap (Fig. 80). The first question to be decided is whether the premaxillary bone should be left *in situ* or should be removed; the answer to this must depend upon circumstances; if the premaxilla is projecting prominently forward it should be removed, but in other cases it may advantageously be left, as its presence serves as a support to the lip and will not interfere with union. The plan of forcibly bending the premaxilla back into position is recommended by some surgeons, but it has the disadvantage, *inter alia*, of pushing the dental border into an abnormal position so that the teeth when cut project backwards.

FIG. 80.—Profile view of a case of double hare-lip. The intermaxillary bone and central incisor teeth are attached to the tip of the nose with which the skin covering is continuous. (Fergusson.)

If it is decided to remove the premaxillary bone the skin and

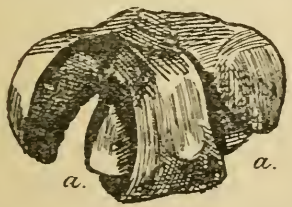

FIG. 81.—Anterior view of the premaxillary bone from a case of double hare-lip. The gum has been divided on each side to show the incisor teeth, *a*, *a*. (Westminster Hospital Museum, No. 392.)

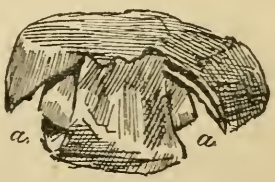

FIG. 82.—Posterior view of Fig. 81.

soft structures on it should be dissected off and carefully preserved and the bone is then cut away with forceps, the gap being at once closed or the operation being completed in two or three weeks. The union of the lip is effected in much the same way as for single clefts, the skin on the premaxillary bone being used to form the columella as shown in Figs. 83, 84, p. 282.

After-treatment.—As respiratory difficulty is sometimes ex-

perienced after the operation, the nurse should not leave the child for three or four hours, and should be directed to draw down the lower lip and jaw if such an event should occur. In cases of single

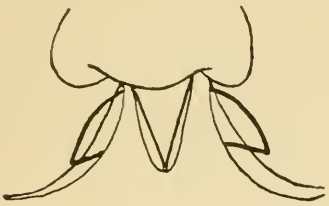

FIG. 83.—Rose's operation for double hare-lip. The dark lines indicate the lines of incision.

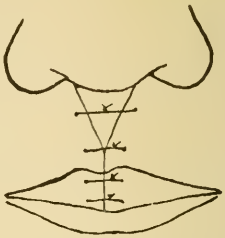

FIG. 84.—Rose's operation completed by suturing of the freshened surfaces.

hare-lip the child may take the breast or bottle, but in double hare-lip it should be fed by means of a tube for the first four or five days.

Cleft of the lower lip is very rare. It is always seen in the middle line, and is due to failure of union of the mandibular plates. The edges of the cleft should be pared and united.

Cleft cheek is dependent upon imperfect union of the maxillary and mandibular plates. The defect is very rare, and is remedied by paring and suturing the edges.

CLEFT PALATE

If the palatine portions of the maxillary plates fail to unite, cleft palate results. The cleft may only affect the uvula, or may extend through the soft and hard palates, along the line of the intermaxillary suture on one or both sides and through the alveolar border, in which case there is associated single or double hare-lip. When the cleft extends through the hard palate the septum nasi is imperfectly developed and may be free, but is usually attached to one side of the cleft, more frequently to the right than to the left. As the child grows the dental arch becomes narrowed, and hence the gap is contracted, but is never completely obliterated.

Cleft palate interferes with sucking, and hence these children require very careful feeding, or they fall into a marasmic condition. Bronchitis and broncho-pneumonia are dangers to be guarded against. In later life the tendency to regurgitation of fluids through the nose and the characteristic defect of speech are well known.

Treatment.—Children with cleft palate must be fed by means of a large teat with a flange of rubber on one side, which will fill up

the gap and allow a vacuum to be formed during sucking. If this plan does not answer, the child must be fed by a spoon or through a tube passed well back to the pharynx while the head is thrown back. It must be carefully protected from cold. The treatment is essentially operative, but in very wide clefts of the hard palate operation does not offer any hope of closure, and in such cases a properly constructed obturator is the only means of treatment. When both hard and soft palates are cleft they should be united at the same operation.

Age for operation.—If the cleft does not involve the hard palate it may be united any time after the first year and should be closed before the child learns to speak, so that faulty intonation may be reduced to a minimum. Clefts of the hard palate may be subjected to operation after the fourth year; but if the cleft is very broad, it is better to wait until about the age of six or seven, for the tissues are then stouter and more likely to unite, the child is intelligent enough to help in the after-treatment, and the cleft has somewhat narrowed, owing to the contraction of the dental arch. No operation is admissible unless the child be in perfect health and free from all throat or ear troubles.

In cases of operation for cleft palate, it is important to defer the operation until the child has been properly trained to refrain from talking, to take food through a tube, and to keep the mouth closed, as he will have to do after the operation; such a plan is of great importance as regards success.

Closure of the soft palate—Staphyloraphy.—The patient

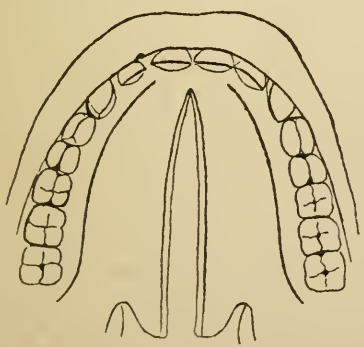

FIG. 85.—Cleft of the hard and soft palates. The line on each side parallel with the teeth indicates that of the incision through which the muco-periosteal flap is raised.

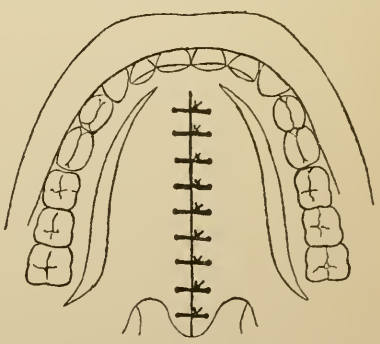

FIG. 86.—Closure of the hard and soft palates. The line seen on each side in Fig. 85 is now a gap which heals by granulation.

is placed under chloroform and the mouth is well opened by Smith's gag; the neck should be over-extended. The uvula being held by

a mouse-tooth forceps, that side of the palate is put on the stretch and a narrow strip is removed from behind forwards; the other side is similarly freshened. Fine silkworm gut and horse-hair sutures are introduced from before backwards about $\frac{1}{6}$ inch apart, and when all have been passed they are tied. Silver-wire is not necessary and is now little used. It is important that the sutures should merely hold the sides of the palate together and should not drag them into apposition, for if there be any tension union will almost certainly fail. If on attempting to approximate the folds it is found there is tension, this must be overcome by making a cut on each side parallel with the cleft but not extending to the free border of the palate; if there is still tension, the tendinous attachments to the hamular process on each side must be separated with a raspatory passed through the lateral incisions; tension may also be overcome by dividing the pillars of the fauces.

Closure of the hard palate—Uranoplasty.—The margins of the cleft are first freshened, and then an incision is made on each side near the alveolar margin extending from before backwards from the anterior end of the cleft to the junction of the soft and hard palates and extending down to the bone (Fig. 85, p. 283). All the soft structures between this incision and the margin of the cleft are then separated from the bone by a curved raspatory, care being taken that the parts are not bruised or unnecessarily damaged. The posterior attachment of the soft palate is now separated from the hard by scissors which are introduced through the lateral incisions. By this means a loose flap is detached from each side, and these are then united from before backwards in the manner described above.

In very wide clefts a broader flap may be secured by separating the mucous membrane from the nasal septum as well as from the hard palate: the septum is usually attached to one side of the cleft and the incision is made some distance up it instead of along the margin of the cleft.

After-treatment.—The food must be of a fluid nature, and should be given by a tube for the first week or ten days; nothing solid which requires mastication should be allowed for at least a fortnight. In severe cases rectal feeding for a few days may be advisable. The child must not be allowed to talk, and examination of the mouth should be deferred until about the tenth day. It is advisable to gently douche the mouth with Condy's fluid or dilute "Listerine" and insufflate a little iodoform once or twice daily.

The sutures need not be removed for three weeks.

When the gap is closed the child should be taught to talk so that its faulty intonation which is partly habit may be improved, and the parents should, before any operation is undertaken, be warned that it will not by any means completely cure the defective speech; unless they are warned of this their disappointment will be very evident.

Deformities of the Limbs

talipes or club-foot

Causes.—Club-foot may be congenital or acquired. The congenital form is sometimes hereditary, and is often bilateral and associated with some other deformity, *e.g.* spina bifida; in some cases it is due to injury at the time of birth.

Congenital cases are probably due in most cases to a faulty position *in utero;* in some to nerve lesions, as in associated spina bifida; in a few to absence or incomplete development of the bones of the leg.

Acquired talipes is the common form and may be due to over-action of one group of muscles, while the opponents act normally; or more usually to the normal contraction of a group against weakened or paralysed opponents; in either case the foot is dragged in that direction towards which the strongest group of muscles act. The most common cause of such disproportionate strength is to be found in infantile paralysis, but it may also be occasioned by hemiplegia, lateral sclerosis, infantile convulsions, or damage to any given nerve trunk. Rupture of a tendon or tendons, cicatricial contraction of muscles following deep-seated cellulitis and suppuration, or the contraction after a severe burn may also induce talipes. Disease or injury of an epiphysis by arresting growth in that bone may also lead to the deformity; thus if growth be arrested at the lower end of the tibia, the fibula becomes in time the longer bone, and the malleolus pressing against the calcaneum may force the foot into the position of T. varus.

Varieties.—The following varieties of talipes are met with, but are by no means of equally common occurrence.

(1) **Talipes equinus** (Fig. 90, p. 288).—The heel is drawn up and the toes are pointed.

(2) **Talipes varus** (Fig. 91, p. 289).—The outer border of the foot rests upon the ground, the sole being turned inwards. These two forms are usually combined, giving rise to

talipes equino-varus (Figs. 92, p. 290, 94, p. 293), which is the most common form of club-foot.

(3) **Talipes calcaneus** (Fig. 87, p. 287) is the reverse of equinus.

(4) **Talipes valgus** (Fig. 97, p. 295) consists in eversion of the foot and is usually associated with pes planus.

(5) **Talipes calcaneo-valgus** is a combination of these two varieties.

(6) **Pes planus** (Fig. 97, p. 295).—The arch of the foot is much flattened, and there is associated T. valgus or genu valgum.

(7) **Pes cavus** (Figs. 95, 96, p. 294).—The arch of the foot is increased, and the toes may be depressed from associated T. equinus (*pes arcuatus* and *plantaris*).

The general anatomy of talipes.—In nearly all forms of talipes the deformity implicates the transverse tarsal arch, but in T. equinus and calcaneus the ankle joint is affected in consequence of the shortening of the ligaments. The degree of wasting of the bones and muscles is proportionate to the severity of the deformity and the uselessness of the foot. In most cases the bones are simply atrophied and there is no gross change in their shape, but the articular surfaces may be partly obliterated or broadened in conformity with the displacement. The contracted muscles are shortened, and their tendons stand out prominently when any forcible attempt is made to correct the deformity; the tendons run in abnormal directions. The weak or paralysed muscles become proportionately elongated, they waste and undergo fatty degeneration and atrophy. The ligamentous structures share in the shortening and contraction or elongation and atrophy in the same way as do the muscles. The skin and subcutaneous structures which are brought in contact with the ground are condensed and thickened as the result of intermittent pressure; corns and bursæ are commonly formed, except in the case of young children who do not put the feet to the ground. In paralytic cases the limb will be found withered, cold, and flabby, and, if the deformity be unilateral, much smaller than that on the sound side.

General plan of treatment.—The mere correction of the deformity is by no means the only object to be aimed at in treatment, for it is essential that the foot must not only look shapely but must work well as a foot. The actual treatment must be prescribed with a full knowledge of the circumstances of the case and with full regard to the degree of the deformity and the amount of

restitution which can be obtained by manipulation. In moderate cases, especially in the young, daily manipulation, cold douching, massage, and galvanism, and in some cases the employment of some form of shoe or mechanical contrivance may effect a cure and should at least be persevered with for a time.

If such means fail, the contracted tendons and fascial structures must be subcutaneously divided, after which the foot may be forcibly wrenched into the correct position. In bad cases more severe operations, such as tarsectomy or excision of particular bones, are needful; and in the worst, even amputation may be necessary to rid the patient of a hopelessly deformed limb, which is not only useless but an encumbrance. In paralytic cases it is obviously essential to encourage by friction, douching, and galvanism the nutrition and functional activity of the paralysed muscles, otherwise a recurrence of the deformity will ensue.

Even when a talipedic foot has been by one or other means placed in its correct position, the patient must wear some form of retentive and supporting apparatus for at least three or four years in order that recurrence may be prevented.

TALIPES CALCANEUS

Talipes calcaneus is a rare deformity and may be congenital or acquired.

Anatomy.—The heel is depressed and the anterior muscles of the leg and anterior ligament of the ankle joint are contracted and shortened. The astragalus is displaced somewhat backwards, and if the tarsal arch is much increased, there will be some degree of displacement at the medio-tarsal joint, the anterior part of the foot being directed downwards, *i.e.* there is associated pes arcuatus (p. 294). In mild and early cases there is no increase in the tarsal arch, the sole remains normal and the toes are not elevated.

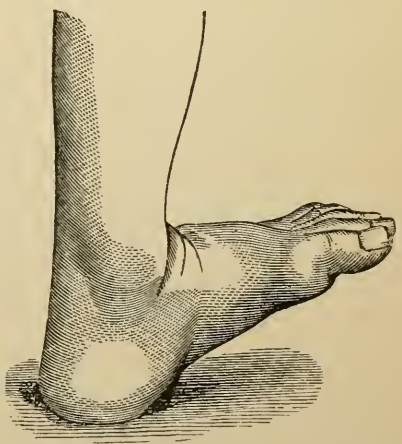

FIG. 87.—Talipes calcaneus (Follin).

The soft structures over the heel are much thickened and a false bursa may develop.

Treatment.—In congenital cases shampooing and manipula-

tion, combined with the use of a malleable iron splint, the angle of which can be altered as improvement is effected, may effect a

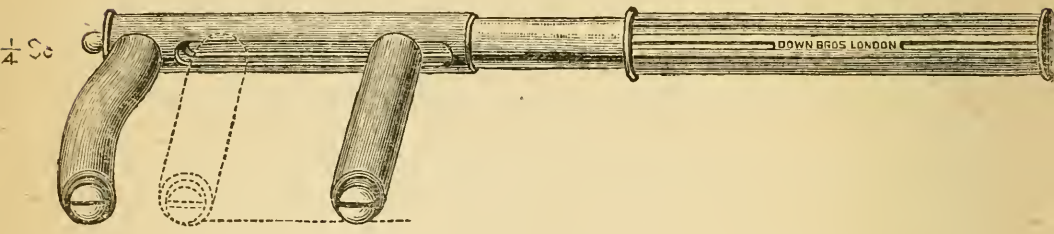

FIG. 88.—Tubby's modification of Thomas's wrench.

cure, otherwise the contracted structures must be divided. The acquired deformity, when slight, may similarly be treated by a mechanical contrivance; Tubby recommends a boot with a toe-depressing spring and a "stop" to prevent the heel dropping (Fig. 89). In marked cases the contracted structures will require division, and the foot may then be forcibly wrenched into position; for this purpose Tubby's modification of Thomas's wrench is the best instrument (Fig. 88).

In paralytic cases the tendo Achillis may be shortened.

FIG. 89.—Walking apparatus for talipes calcaneus with toe-depressing spring (Tubby).

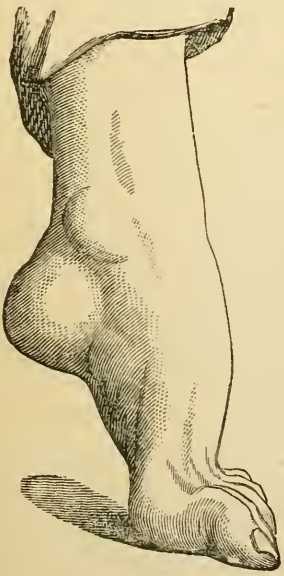

FIG. 90.—Talipes equinus (Follin).

TALIPES EQUINUS

Talipes equinus is practically always an acquired deformity and varies much in degree. The position of T. equinus is sometimes voluntarily assumed by patients with a shortened limb in order to bring the foot to the ground. Normally the foot can be dorsiflexed to an angle of about 18°; in T. equinus this range of flexion is diminished. In the mildest cases the foot can be placed at a right angle with the leg; in the worst it is fully extended, and the dorsal aspect of the toes comes

in contact with the ground so that walking is impossible if the deformity is bilateral; such an extreme degree is, however, rare, and in the majority of cases the following condition will be found to exist.

Anatomy.—The heel is raised, owing to contraction of the tendo Achillis, so that the patient walks on the heads of the metatarsal bones. This position leads to broadening of the anterior part of the foot and to induration of the skin and subcutaneous tissue, with the formation of painful corns in this situation. The degree of the deformity is most marked when the knee joint is extended. The head of the astragalus is necessarily rendered prominent, and there may be some degree of subluxation at the astragalo-scaphoid joint. The tarsal arch may be considerably increased, the fascial structures and plantar ligaments being shortened and the dorsal stretched. The posterior ligament of the ankle joint is shortened, and the peroneus brevis and longus and flexor tendons may be contracted; if the arch of the foot is much increased, the short plantar muscles are also contracted.

Treatment.—In slight degrees manipulation and moderate exercise, combined with galvanism to the muscles in paralytic cases, will usually cure the deformity.

When this is more marked, the tendo Achillis must be divided, and the plantar fascia may require similar treatment. After tenotomy the limb should be at once put up in plaster of Paris or in a Scarpa's shoe until healing is complete.

TALIPES VALGUS

Talipes valgus is nearly always met with in association with pes planus (Fig. 97, p. 295), and is sometimes due to absence of the fibula. The foot is everted, and the arch is flattened, owing to yielding at the medio-tarsal articulation. The peronei muscles are contracted and shortened.

Treatment.—If manipulation and properly constructed boots (see p. 296) do not correct the deformity the peronei must be divided.

TALIPES VARUS

Pure talipes varus is extremely rare, but may be due to paralysis of the peronei. The foot is adducted to the middle line and is inverted at the medio-tarsal joint; the toes are turned inwards, the

plantar fascia is contracted, and the arch of the foot is increased. The patient walks on the outer border of the foot.

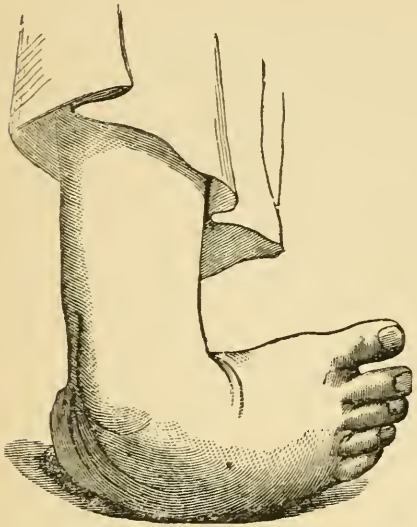

FIG. 91.—Talipes varus (Follin).

Treatment. — The treatment of the condition is similar to that recommended for talipes equino-varus.

CONGENITAL TALIPES EQUINO-VARUS

This deformity is more common in boys than girls, is usually bilateral, and is sometimes associated with other deformities, such as spina bifida. It varies considerably in degree. In the mildest cases it can be reduced by manipulation, but the foot assumes the unnatural position as soon as it is released. In the next degree complete restoration to the normal cannot be effected by this means, and in the worst form the foot is rigidly fixed in the abnormal position.

Anatomy.—The foot is extended at the ankle joint and the heel is drawn up; the sole is inverted, the toes are adducted to the middle line of the body, the inner border of the foot is raised, and the foot is folded on itself at the medio-tarsal joint. The great toe is flexed, and often considerably adducted, so that the interdigital space is much broadened. The internal malleolus is less, and the external more prominent than usual. If the child is old enough to have walked, the deformity is accentuated, and as the outer border of the foot (and in bad cases part of the dorsum also) comes in contact with the ground, the skin and subcutaneous tissue

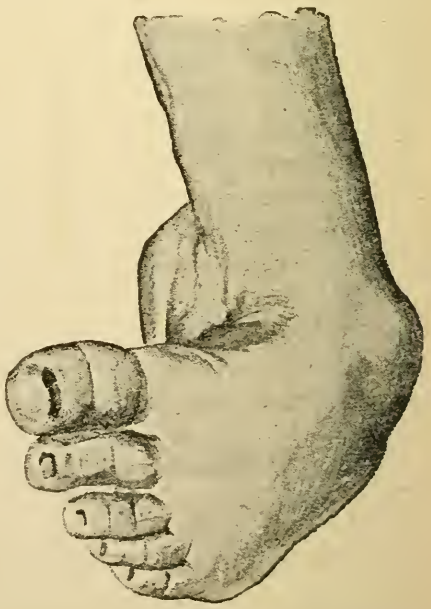

FIG. 92.—Congenital talipes equino-varus (Tubby).

are much thickened and hypertrophied, and false bursæ and corns form, which cause considerable pain, and may, on account of inflammation, make walking impossible. In the sole of the foot there is a transverse crease opposite the medio-tarsal joint, and from this a longitudinal crease runs forwards to the first interdigital space. These creases are diagnostic of the congenital nature of the deformity. The tarsal bones are altered in shape. Owing to curvation of its neck, the head of the astragalus forms a marked dorsal prominence and is directed downwards and inwards. There may be partial luxation at the mid-tarsal joint. The osseous deformity becomes more marked as growth proceeds and when the child begins to walk; as ossification advances, the deformity may be rendered permanent. The anterior tibial muscles are deflected inwards, and those behind the inner ankle forwards; the peroneus longus tendon grooves the os calcis instead of the cuboid. All the muscles remain undeveloped and tend to undergo atrophy. The dorsal ligaments, and those on the outer border of the foot, are lengthened and attenuated, while those on the inner side and the plantar fascia are contracted.

Owing to the nature of the deformity the child has a peculiar waddling gait, and in marked cases has to lift one foot over the other (*reel-feet*).

Treatment.—The earlier treatment is commenced the better, its nature depending upon the degree of deformity and the age of the patient.

In the least severe cases, when the deformity can be overcome by manipulation, this, combined with douching, massage, and galvanism to the muscles, should be conscientiously carried out two or three times a day. Manipulation must be gentle and steady, and the foot must be everted and adducted at the mid-tarsal joint, and also flexed at the ankle joint. During the treatment a flexible metal splint, or tin shoe with a movable foot-piece, should be worn, and must be moulded to fit the outer side of the leg and foot to correct the varus, and to the posterior surface of the leg and sole of the foot to correct the equinus. As the position of the foot is improved, the flexible metal can be easily adapted to the shape of the limb. This treatment, if carefully persevered with, will correct the deformity; but

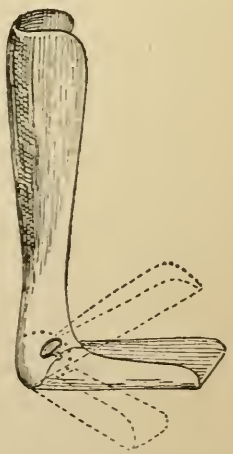

FIG. 93.—Tin shoe with quadrant movement at heel (Tubby).

the retentive apparatus must be worn for many months to prevent recurrence.

In more severe cases, in which the deformity cannot be wholly reduced by manipulation, tenotomy should be performed, and the operation may be undertaken when the child is only a few days old. It is advisable to first correct the varus by dividing the tibialis posticus, flexor longus digitorum, and, if necessary, the tibialis anticus and extensor proprius hallucis tendons, together with the plantar fascia. At a later date the equinus may be overcome by division of the tendo Achillis. Tenotomy and fasciotomy may be followed by manual or instrumental wrenching in order to correct the deformity. After tenotomy, the foot may be immediately placed in a plaster casing; but if the normal position cannot be at once attained, it is better practice to effect gradual reduction, so that the bones may gradually be moulded to the proper position. For this purpose Scarpa's or Little's shoe is the best apparatus. The gradual treatment must be conducted over a period of about two months to overcome the varus, and for another like period after the tendo Achillis has been divided. When the normal or best position has been attained by these means, the patient should wear a retentive walking apparatus for three or four years, or more, in order to prevent relapse. During the first year, it is also advisable to wear a light apparatus at night.

In the worst cases the above treatment, with forcible attempts at rectifying the deformity by means of the wrench, may give a good result, provided plenty of time and patience be expended; but should it fail, some further operative measures will be needed.

Tarsotomy and tarsectomy.—These operations are but rarely called for, and should never be performed on young children, for in them milder means will overcome the deformity. Of their value the opinions of surgeons best qualified to judge vary within the widest limits; some extol these operations, while others decry them as almost unjustifiable. It seems, however, that the truth lies between the two extremes; there are certainly cases in which no other treatment can be effective, and recourse must be made to some such form of operation imperfect though its results may be.

Of the many operations which have been devised, the removal of the astragalus or the division of its neck, or the removal of a wedge-shaped portion of the tarsus (the base being at the outer border of the foot), are those of the greatest value, that one being given the preference which seems most suitable to the case. It must not be forgotten that such operations are by no means free

from danger, especially in the hands of those not well acquainted with the details of operative surgery, and that even the most brilliant result leaves much to be desired. While it is quite true that a shapely foot, the sole of which comes upon the ground, may be secured, yet such a foot is better in appearance than in fact, for it has lost many of the characters—and those by no means the least important—of the normal foot. Thus the plantar arch is more or less destroyed, and there is impairment or even complete fixity at the ankle and mid-tarsal joints, so that the foot has lost one of its most characteristic and valuable features—elasticity. At the same time, it is only fair to state that such operations certainly shorten the time taken up in cure, and are less liable to be followed by relapse; but should such unfortunately occur, nothing further can be done, and amputation is the only means of ridding the patient of a deformed, useless, and often painful foot. Amputation is, however, very rarely needed, and should usually be reserved for those cases in which suppuration occurs in the bursæ and thickened tissues as the result of prolonged pressure and irritation.

ACQUIRED TALIPES EQUINO-VARUS

The acquired deformity is nearly always due to infantile paralysis (Fig. 94) affecting the peronei and muscles on the front of the leg, although the tibialis anticus may escape. In some cases the deformity is spastic, and in a few is dependent upon injury or disease about the ankle joint or tibial epiphysis.

FIG. 94.—Talipes equino-varus from infantile paralysis in a child aged four and a half years (Tubby).

The deformity is much the same as that of congenital origin (Fig. 92, p. 290); but the heel is often, especially in spastic cases, more raised, the transverse and longitudinal creases on the sole are wanting, and the head of the astragalus is not directed inwards.

The paralysed muscles are fatty and atrophied, while the others are tensely contracted.

Treatment.—In paralytic cases the treatment must have reference to the nutrition and improvement of the paralysed muscles, and the rectification of the deformity. These aims may be carried into effect by douching, friction, galvanism, and manipulation, combined with the use of a tin shoe or malleable splint

(Fig. 93, p. 291), as recommended for the congenital deformity. Tenotomy must be performed if needful.

TALIPES CAVUS

Talipes cavus is usually an acquired deformity due to weakness of the anterior muscles of the leg, and is often associated with slight talipes equinus.

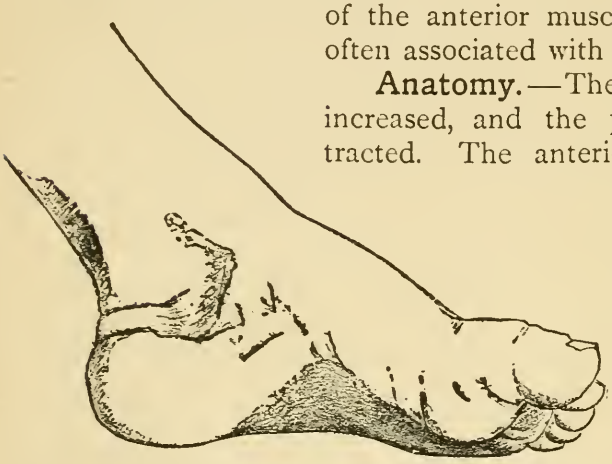

FIG. 95.—Talipes arcuatus in a boy aged five and a half years (Tubby).

Anatomy.—The arch of the foot is increased, and the plantar fascia is contracted. The anterior tibial muscles are tense, so that the metatarso-phalangeal joints are over-extended, while the inter-phalangeal joints are flexed. The tendo Achillis is slightly contracted. According to some the condition is primarily due to paralysis of the lumbricales, interossei, and short flexors of the great toe; while others consider that it is a compensatory change to slight T. equinus, the arch being increased so that the heel may come to the ground. If the balls of the toes are on the same level as the heel the condition is spoken of as *T. arcuatus* (Fig. 95), but when they are depressed to a lower level as *T. plantaris* (Fig. 96). There is considerable inconvenience in walking, and often pain, owing to the formation of corns on the sole opposite the heads of the metatarsal bones—especially the first and fifth.

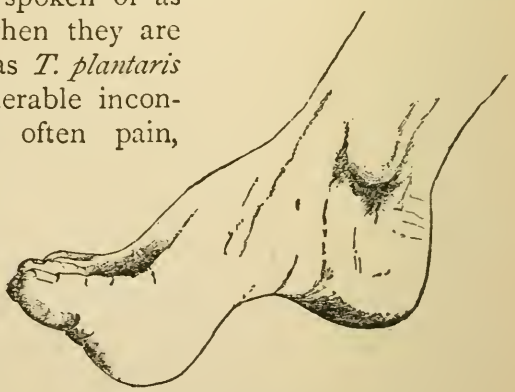

FIG. 96.—Talipes plantaris (Tubby).

Treatment. — The equinus must be corrected by division of the tendo Achillis; the plantar fascia and extensor tendons will also need division. The foot should then be carefully put up on a back splint with a foot-piece, and the

position maintained for a month or more, daily manipulation being employed after ten days. The treatment is sometimes disappointing, especially in unskilled hands, and a second operation may be necessary.

TALIPES CALCANEO-VALGUS

Talipes calcaneo-valgus is a rare form of club-foot, and may be congenital or acquired as the result of infantile paralysis.

Anatomy.—The heel is depressed, and the outer border of the foot is everted and raised, the part in front of the mid-tarsal joint being abducted. The sole is flattened. The anterior tibial and peroneal muscles are contracted, and the ligaments on the outer side of the foot are shortened.

Treatment.—Rectification of the deformity may be effected by manipulation, douching, etc., combined, if needful, with division of the contracted tendons.

PES PLANUS—FLAT OR SPLAY FOOT

Pes planus is usually associated with more or less T. valgus and is *congenital* or *acquired.* In the latter case the condition may be due to rickets, and is often associated with genu valgum. Flat foot often makes its appearance, or at least increases in severity, about the age of puberty—that is, at a time when growth is rapid, and the naturally weak arch may yield from laxity of its

FIG. 97.—Pronounced flat foot (Tubby).

ligamentous supports owing to the rapidly increasing weight of the body. This is especially likely to occur in those who are naturally

weak and feeble, who are recovering from some severe illness, or whose occupation (*e.g.* waiters) entails long hours of standing.

Anatomy.—Owing to the loss of the longitudinal and transverse arches the foot becomes proportionately lengthened and broadened; the inner border is convex, thicker than natural, and rests upon the ground, and the portion of the foot in front of the mid-tarsal joint is everted and abducted (Fig. 97, p. 295). The inferior calcaneo-scaphoid ligament, on which the head of the astragalus rests, is stretched and the plantar fascia yields, so that the head of the bone and the scaphoid are displaced downwards and in bad cases may rest upon the ground. The tibialis posticus tendon is weakened, and the anterior and posterior leg muscles tend to waste, while the peronei are contracted and often in a state of spasm.

The patient's gait is shuffling and shambling, and progression is slow; fatigue is soon induced, and there is considerable pain, especially beneath the astragalo-scaphoid joint. The foot sweats easily and becomes puffy at night, especially if, as is often the case, the veins are congested or varicose.

Treatment.—Complete rest for two or three weeks is essential; it alleviates the pain and diminishes the spasm of the peronei muscles; during this time shampooing, cold douching, and manipulation will do much good.

In mild cases the patient should have a Whitman's valgus sole-pad fitted to the boots, which raises the inner border and helps to sustain the weakened arch, which may be further strengthened by directing the patient to raise himself on to the tips of his toes as a means of exercise, and should be employed two or three times daily for as long a time as the strength of the muscles will permit without pain or fatigue. If it be found that in spite of rest the peronei remain contracted the tendons should be divided, and gradual replacement may be then effected by the use of Scarpa's shoe and daily manipulation. Wrenching the foot under an anæsthetic and confinement in a plaster casing for three or four weeks, followed by daily manipulation, is often serviceable.

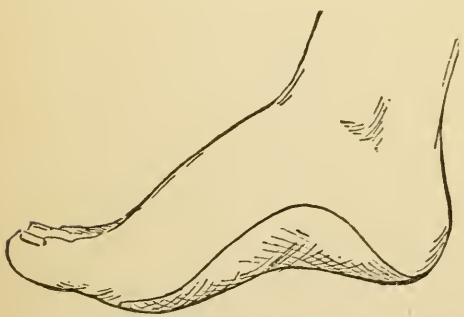

FIG. 98.—Whitman's valgus sole-pad applied (Tubby).

In very bad cases, where no good can be effected owing to

arthritic changes, the astragalo-scaphoid joint may be excised, and the bony surfaces united by suturing or pegging after the foot has been placed in a good position by forcible manipulation; it is then put up in plaster, and the patient is not allowed to put it to the ground for three or four months, *i.e.* until the ankylosis between the bones is sound enough to sustain the necessary weight. Such treatment is, however, very tedious, and few patients can afford the time.

TENOTOMY

The division of tendons, ligaments, and tense bands of fasciæ may be necessary for the correction of congenital or acquired deformities. The operation may be performed by the *open* or *subcutaneous* method; the former should always be used in the case of the sterno-mastoid in view of its dangerous relations, and may also be employed for the biceps femoris if the surgeon feels uneasy about the peroneal nerve; with due antiseptic precautions the open method is as safe as the subcutaneous.

The subcutaneous operation.—The subcutaneous operation may be performed in two ways: (1) by cutting the tendon from within out; or (2) from without in; in the first method the knife is placed beneath the tendon, and in the second between it and the skin; the method to be selected depends upon the situation of the tendon. The patient is anæsthetised, and the part, *e.g.* the foot, is held by an assistant in such a position as will relax the tendon to be divided; the surgeon then passes the narrow-bladed sharp-pointed tenotome deep or superficial to the tendon according to the method he adopts. This is withdrawn, and replaced by the blunt-pointed instrument. The knife is passed on the flat, and the cutting edge is then turned towards the tendon which is divided by slight sawing cuts, while the assistant steadily draws the foot into that position which renders the tendon tense. When the division is completed the tendon parts with a sudden snap; any tense bands which can be felt should be divided, but in the case of the biceps femoris this should never be done, a tense band will always be felt—it is the peroneal nerve. The small skin puncture is closed with a pad of wool and collodion.

Rectification of the position.—After tenotomy or fasciotomy the abnormal position necessitating the operation may be at once completely rectified, or adjustment may be postponed for a week or ten days, or may be gradually effected. As a rule, it may be said

that gradual reposition is the best, and should always be carried out if the tendon on division is separated by a gap of an inch or more, if the open method has been resorted to, or if a vessel or nerve of importance has been damaged.

Dangers of the operation.—In passing the knife care must be taken that the tendon is not transfixed, or it will only be partially divided. Should this happen the knife must be re-introduced.

If an artery be wounded, as may occur to the posterior tibial during division of the tibialis posticus and flexor longus digitorum tendons, the accident will be recognised by a sudden spurt of blood by the side of the knife and by blanching of the foot. Under such circumstances it is necessary to apply a pad and bandage, and postpone all attempts at reposition of the foot for about a fortnight. Division of a nerve would necessitate laying open the wound, and thus converting the subcutaneous into an open operation, in order that immediate suture may be performed.

Suppuration after tenotomy ought never to occur if due precautions have been taken.

Non-union of a tendon may result if suppuration follows the operation, or if the separation between the cut ends is great, and has been further increased by immediate reduction of the deformity.

Tenotomy of the tibialis anticus tendon is usually performed just before the tendon reaches the internal cuneiform bone. The knife is passed beneath the tendon from the outer side, so as to avoid the anterior tibial artery.

Tenotomy of the extensors of the toes may be performed on the individual tendons about a finger's breadth[1] behind the heads of the metatarsal bones; the knife may safely be passed superficial to the tendon, which is divided while the toes are forcibly flexed.

Tenotomy of the tibialis posticus and flexor longus digitorum.—The leg is placed on the outer side, and the knife is passed between the tendons and the bone about two fingers' breadth above the small tubercle which can be felt near the base of the internal malleolus. Care must be taken that the knife is not thrust too deeply, or the posterior tibial artery may be wounded.

The tendo Achillis is divided just above its insertion, at its narrowest part. The knife may be passed superficial to the tendon which is divided inwards; there is practically no risk of wounding the posterior tibial artery if care is taken, but owing to its proximity

[1] It must be remembered that when distance is measured by fingers' breadth, the finger must be that of the patient, not of the operator.

many surgeons prefer to divide the tendon from its deep surface.

The peroneus longus and brevis are cut about three fingers' breadth above the base of the external malleolus, the knife being passed between the tendons and the bone.

The biceps femoris is best divided by the open method, so that all danger of wounding the peroneal nerve, which lies just internal to it, is avoided. If the subcutaneous method is employed the knife is passed to the inner side of the tendon, *i.e.* between it and the nerve, and the tendon is divided outwards; when the division is complete the nerve will be felt as a tense rounded cord.

The semi-membranosus and semi-tendinosus should be divided from the deep surface just above the internal condyle.

The sterno-mastoid must be cut by the open method (see p. 277).

FASCIOTOMY

Fasciotomy or division of tense bands of fascia is necessitated for the cure of Dupuytren's contraction of the fingers (p. 307), and for some forms of club-foot.

The plantar fascia is best divided by passing the knife beneath the skin and superficial to the fascia. The point of division and the number of punctures for the division of individual bands of fascia must be determined on the merits of the case; sometimes numerous punctures are necessary. Complete removal of the fascia by the open method is recommended by some surgeons.

SYNDESMOTOMY

Syndesmotomy or division of ligaments is occasionally necessary in cases of club-foot; Parker's astragalo-scaphoid capsule (formed by blending of the ligaments) sometimes requires division in cases of unyielding talipes equino-varus. This capsule can be divided, in association with the tibial tendons, at a point just in front of and on a slightly lower level than the tip of the internal malleolus. The knife is entered beneath the skin, and cuts all structures down to the bone.

GENU VALGUM

Varieties and causation.—Genu valgum may be rachitic, static, or pathological. **The rachitic form** occurs in young children,

and is due either to bending of the soft rickety bones, the femur inwards and the tibia outwards, or to unequal growth at the epiphysary line, leading to disproportionate length of the internal condyle, either because this has grown inordinately, or because the external condyle has ceased to develop from premature ossification of its epiphysary line.

Static genu valgum becomes manifest at or about the age of puberty, and is sometimes predisposed to by early rachitic changes, but may occur quite independently of that disease. It is dependent upon the habitual assumption of that position of rest in which the main weight of the body is thrown on the internal lateral ligament and the sole of the foot, and hence these, being unable to bear the long-continued strain, eventually yield; the knee goes inwards and the arch of the foot is flattened (pes planus). The associated flat foot aggravates the genu valgum, which is still further increased and rendered permanent by the fact that the internal condyle, being to some extent relieved of pressure, grows more quickly than the external, and becomes elongated. After a time the external lateral ligament, the biceps tendon, and the ilio-tibial band become somewhat shortened and contracted. The tibia is rotated outwards, and the patella is more or less displaced to the same side.

The pathological causes of genu valgum must be sought in inflammation of or injury to the epiphyses, in infantile paralysis, or fracture of the lower end of the femur or upper end of the tibia; genu valgum from these causes is rare and usually slight in degree.

Signs.—There is, especially in women, a natural tendency to knock-knee in consequence of the breadth of the pelvis. Slight degrees are but little noticeable, but when the condition is marked the patient walks with an awkward, rolling, shambling gait, progresses slowly, and may knock together or cross the knees, so that, when young, he not infrequently falls. Fatigue is easily induced, and there is often considerable aching in the legs after exertion or at night. To estimate the degree of deformity the patient must be directed to stand up with the knees about half an inch apart; in this position the malleoli should, if the legs be normal, be in contact, and hence the degree of genu valgum can be estimated by the distance they are separated. If the legs be flexed the deformity disappears probably because the posterior surfaces of the condyles of the femur are but little affected, and hence at this point the axis of articulation is practically normal.

Treatment.—The rachitic form may, as the initial disease

becomes cured under treatment, considerably improve, especially if manipulation be properly carried out; indeed, in slight cases the child may eventually show no signs of the deformity. In addition to the dietary and tonic treatment indicated by the rickets (chap. v. vol. iii.), the child should not be allowed to walk, and if he cannot be kept quiet otherwise an outside splint reaching below the sole must be put on each leg, so that it is impossible for him to stand; such a splint, moreover, may be used to correct the deformity, a pad passing round the inner side of the knee being used to gradually draw it outwards. In slight cases no mechanical appliances are

FIG. 99.—Genu valgum (Follin, after Macewen).

needful, but night and morning, after cold douching and massage to the limb, gentle manipulation should be employed with the view of correcting the deformity; the force used must be moderate, and should not be sufficient to cause pain. In some cases it may be advisable to divide the shortened and contracted tendinous and fascial bands on the outer side of the joint.

The static form must be rectified by correcting the faulty habit of standing which occasions it, and also by treating the pes planus.

Operation becomes necessary if the deformity cannot be overcome by manipulation and the measures above indicated, and is of such a degree as to be noticeable or inconvenient. In cases where the elongation of the inner condyle, and the bending of the femur

and tibia is pronounced, operation will be required. Operative interference is also indicated in children over four or five years of age when no further treatment by other means is likely to prove beneficial.

Osteotomy.—Of the various operations for osteotomy in cases of genu valgum that devised by Macewen is most in favour. It is thus performed:—

A small incision is made down to the bone just above and a little in front of the adductor tubercle on the internal condyle of the femur; the chisel is then introduced, and is turned at right angles to the shaft of the femur, the leg resting on its outer side on a sand-bag. By a few strokes of the mallet the inner and anterior two-thirds of the bone are divided, and the remainder is broken by steadily bending the extended limb to the inner side. The superficial wound is closed, and the limb fixed on a back splint for ten days, when the wound will be found soundly healed; the limb must now be put up in a plaster casing, which may be removed in about six weeks, and the patient be allowed to get about on crutches. If the tibia is much curved it may be necessary to divide it just below the epiphysary line if the osteotomy of the femur does not correct the deformity; the contracted structures on the outer side may also need division.

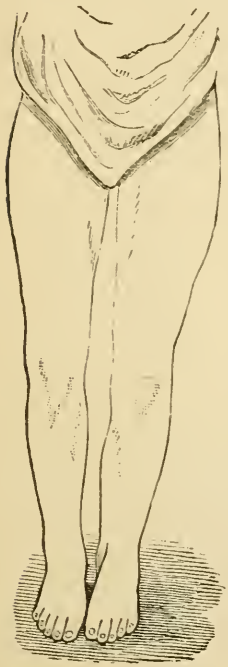

FIG. 100.—The same case as Fig. 99, p. 301, after osteotomy.

Sir William MacCormac recommends that the osteotomy should be made from the outer side, about three fingers' breadth above the patella; the operation is conducted as already described, and the after-treatment is the same. The advantages claimed for this procedure are that the bone is divided at its narrowest part and at some distance from the epiphysary line.

Ogston's operation consists in making a small wound just above the inner condyle, and then introducing a saw obliquely in front of the internal condyle to the intercondylar notch; the condyle is then sawn off obliquely, and as the leg is drawn into place it slips further upwards. This method is useful when the elongation of the condyle is the chief anatomical defect, but it has the disadvantage of opening the knee joint, and is now rarely performed.

BOW-LEGS

In bow-legs there is a curvature of the tibiæ and fibulæ outwards and forwards which is commonly the result of rickets. The curvature is usually most marked in the lower half of the legs.

Treatment.—Rickety children should be prevented standing so that the soft bones may not yield. When the bones are sufficiently pliable the deformity may be overcome by careful manipulation and a splint may be placed on the inner side of the leg, *i.e.* on the concave side of the curve. In bad cases when the child is over four or five years of age and no hope of cure can be expected by the above means osteotomy of the tibia should be performed. An incision is made along the crest of the tibia, and the saw is then introduced to its inner side; when the bone has been divided the fibula is broken and the deformity forcibly reduced. The wound must be antiseptically dressed and the limb placed on a back splint with a foot-piece for ten days, when the wound will be found healed; a plaster casing must be applied for another three or four weeks to allow firm union of the bones to occur.

GENU VARUM—BANDY-LEGS

Genu varum is dependent on rickets and consists in an outward bend of the legs due to curving of the lower end of the femur, the upper ends of the tibia and fibula and the knee joint. The external condyle of the femur is longer, and in the internal shorter than natural, and the ligamentous and tendinous structures on the inner side of the joint may be shortened. The treatment is conducted on the same lines as for genu valgum.

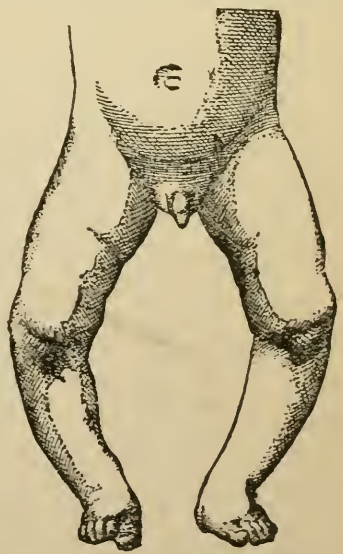

FIG. 101.—Genu varum of rachitic origin (Tubby, after Rédard).

GENU RECURVATUM

Over-extension at the knee joint may be a congenital condition or may result from rickets, infantile paralysis, or after a bad excision of the knee joint. A slight degree is common, but marked deformity rare. If it cannot be remedied by manipulation and an anterior

splint, division of the tense lateral ligaments may be required. In bad cases resection of the joint may be undertaken.

HALLUX VALGUS

Hallux valgus is more frequent among women than men, and is usually due to wearing short and pointed boots which crowd the toes together and deflect the great toe towards the middle line of the foot. The displaced toe rides over or under the second, and in the former case there may be associated hammer-toe. The head of the metatarsal bone and base of the first phalanx form a prominence on the inner side of the foot which is covered by a bursa and dense hard skin which may be the seat of a painful corn. The bursa may inflame and suppurate and lead to disease of the bone beneath or to destructive arthritis (Fig. 102).

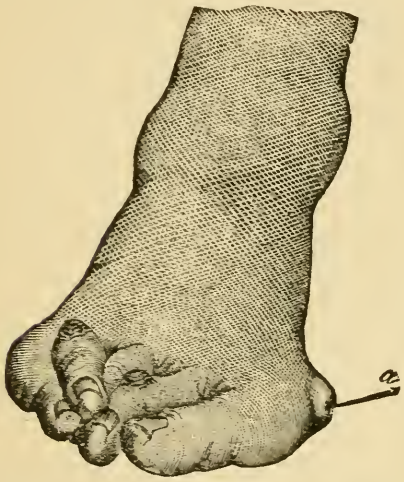

FIG. 102. — Hallux valgus with a bursa over the metacarpo-phalangeal joint. The bursa has inflamed and burst (*a*). (Follin.)

The ligaments and muscles on the inner side of the toe are stretched and lengthened, and those on the outer side proportionately shortened; the extensor proprius hallucis is deflected towards the middle line, and is rendered tense, so that it tends to increase or at least maintain the deformity. Hallux valgus may be associated with osteo-arthritis, which increases the deformity. The condition is usually bilateral, although worse on one side than the other. The patient complains of pain in walking and consequent lameness which becomes much more marked if the bunion inflames.

Treatment.—In slight cases the deformity may be rectified if the patient will wear properly-made boots. Digitated socks are also to be recommended, and some advise the use of a toe-post between the first and second toes. Cold douching and manipulation should be employed night and morning. In bad cases an operation must be undertaken. A very successful procedure is as follows:—

The bursa is carefully dissected away, the tendon of the extensor proprius hallucis is divided, and the head of the metatarsal bone is removed. Some surgeons advise resection of the joint or

simple division of the neck of the metatarsal bone. The wound must be aseptically dressed and the toe kept in its proper position (*i.e.* the inner border in a line with that of the foot) by a splint.

HALLUX RIGIDUS—HALLUX DOLOROSUS

Hallux rigidus consists in flexion of the toe at the metatarso-phalangeal joint. It usually occurs in men who wear short and stiff boots, and is often associated with flat foot. The head of the metatarsal bone projects towards the sole and a bursa may form over it. There is considerable pain, especially if any attempt is made to extend the toe; rigidity is usually due to matting of the tendons and periarticular structures as the result of inflammation, but it is by no means constantly present.

The **treatment** consists in wearing proper boots and correcting the flat foot; if this proves ineffectual, the head of the metatarsal bone should be removed.

INGROWING TOE-NAIL

This painful condition is usually met with in those who wear tight boots, and may be brought about by cutting the nail short and square so that a sharp angle is left which irritates the soft structures. These inflame and ulcerate, and a foul discharge escapes from beneath the granulating edge which grows up over the nail. The condition nearly always affects the great toe, and is frequently bilateral. Somewhat similar trouble is sometimes met with affecting the fingers. The nail is broad, and inflammation of a very intractable nature occurs about its bed; the end of the finger becomes clubbed, inflamed, ulcerated, and tender (onychia "maligna," Fig. 104).

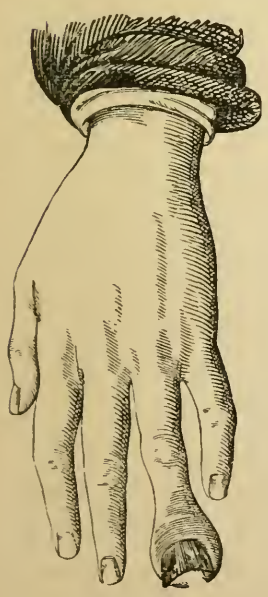

FIG. 104. Onychia "maligna" (Fergusson).

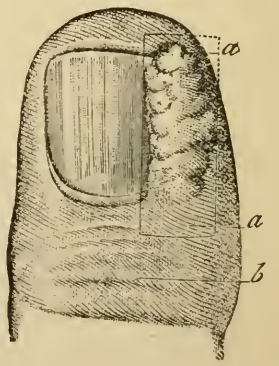

FIG. 103. — Ingrowing toe-nail. *a*, the area of inflammation of the soft structures; *b*, line of the interphalangeal articulation. (Follin.)

Treatment.—Slight cases of ingrowing toe-nail may be much relieved if the nail be well pared down at the side and the surface scraped so that it is made thinner. In troublesome cases half the nail should be removed under gas. A director is pushed beneath it and it is slit up, and the half on the affected side is then torn out with forceps. The granulating area at the side should be cut away and a gauze dressing be applied for a few days. Onychia of the fingers requires similar treatment.

HAMMER-TOE

Hammer-toe usually affects the second toe and is bilateral; it may be congenital and seems to be hereditary. It is sometimes associated with and caused by hallux valgus, the great toe riding over the second.

The metatarso-phalangeal joint is hyper-extended (dorsi-flexed), the second phalanx is flexed, and the third is usually in a line with the second; the head of the metatarsal bone is prominent in the sole. On the dorsal aspect of the first interphalangeal joint the skin is thickened and is often the seat of a painful corn, beneath which is a bursa; the end of the toe coming in contact with the ground is broadened and flattened out, and a corn is present which causes the pain and lameness of which the patient complains. The lateral and glenoid ligaments are shortened and thus keep the toe in its abnormal position; after a time the tendons are also contracted.

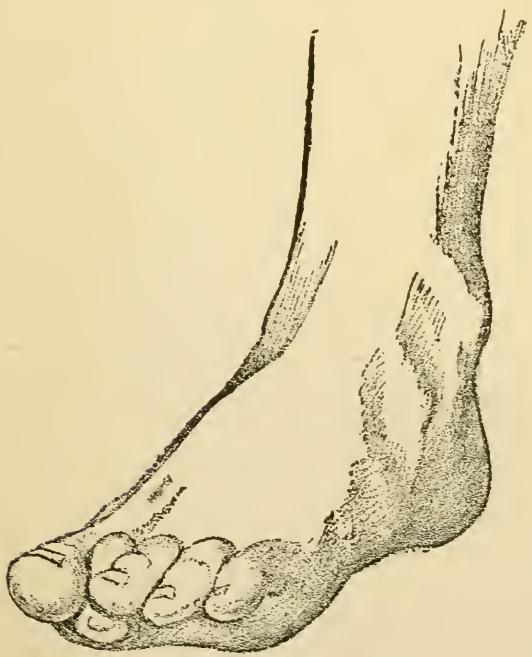

FIG. 105.—Hammer-toe (Tubby).

Treatment.—In slight cases daily manipulation and wearing proper boots will overcome this painful deformity. In bad cases

operation is required. A fine fascia knife is inserted in the middle line of the plantar aspect opposite the first interphalangeal joint, and being turned to either side divides the contracted flexor tendons and the lateral ligaments; care must be taken to avoid the digital vessels and nerves if possible. When the toe has been straightened it must be kept in its proper position by a splint. Other procedures consist in excision of the head of the first phalanx or excision of the joint; amputation is rarely necessary, but is often resorted to as a speedy method of giving relief.

WEBBED FINGERS AND TOES—SYNDACTYLISM

The fingers are more frequently webbed than are the toes; two or more may be united. The bond of union usually consists of skin and subcutaneous tissue only, but the tendons may also be fused; in the worst cases (and these are not amenable to operation) the bones are also joined.

The web may be short or extend the whole length of the digit.

The deformity may be corrected by dissecting up two flaps—one from the dorsal, and the other from the palmar aspect in opposite directions; the fingers are thus separated and the flaps, which must be carefully planned so that no raw surface is left, are stitched in position.

SUPERNUMERARY FINGERS AND TOES—POLYDACTYLISM

Supernumerary fingers and toes are usually imperfectly developed, and may have no skeleton. They are often hereditary. Amputation should be performed.

DUPUYTREN'S CONTRACTION

Dupuytren's contraction is more common in males than females, and usually occurs after forty-five years of age. The tendency to its formation is in some cases hereditary, and seems to be associated with gout and rheumatism. Repeated slight traumation is an etiological factor, the contraction being often seen in those who use tools which press upon the palm, *e.g.* carpenters and engravers. W. Anderson suggests that the irritant causing the contraction is of a parasitic nature, and others consider that it is of nervous origin.

Morbid anatomy.—The ring and little fingers are especially

affected, the contraction usually beginning in the former. The tendons are not in any way affected or implicated, the contraction being limited to the palmar fascia, the digital slips of which send lateral prolongations downwards which are attached to the heads of the metacarpal bones and the sides of the phalanges. The fascia is contracted and hypertrophied, and definite fibrous nodules of small size may be felt in it; the skin becomes dimpled, puckered, thickened, and contracted, and blends with the fascia beneath. The palmar fat atrophies. The contracted bands of fascia may be easily felt, and are seen as rounded cords in the palm. As the contraction is in progress the movements of the fingers become less free and the patient complains of increasing stiffness; the two proximal phalanges are flexed towards the palm, but the distal one remains in a line with the second. The condition increases slowly but certainly, and ultimately the rigidly flexed fingers very much impair the usefulness of the hand.

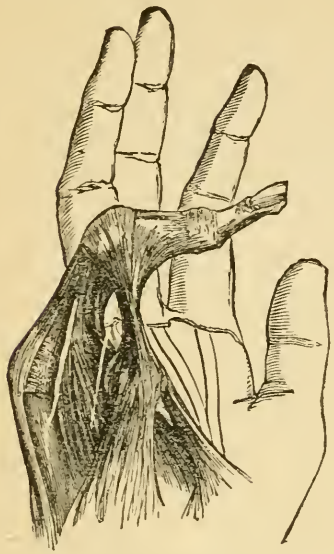

FIG. 106.—A dissection illustrating the contraction of the palmar fascia and its prolongations in Dupuytren's contraction (Tubby, after Druitt).

Treatment.—In the very early stages the patient should be directed to counteract the tendency to contraction by manipulation and massage of the palm, but when the flexion is established operation is the only means of effecting a cure. The contracted fascia should be divided by Adams's method, viz. in the palm on each side of the puckered portion of skin, and also the lateral prolongations passing to the sides of the phalanges; care being taken not to wound the digital vessels and nerves. Some surgeons prefer to divide the bands or dissect them out by the open method. When the finger has been straightened it should be kept in position by a palmar splint.

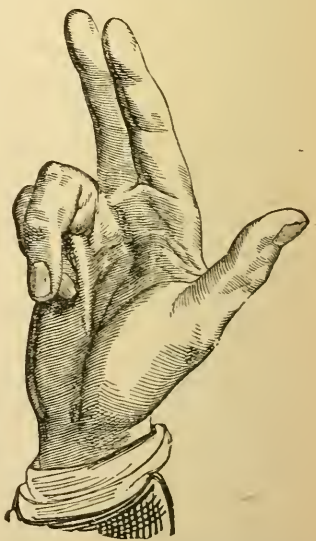

FIG. 107.—Dupuytren's contraction (Fergusson).

CONGENITAL CONTRACTION OF THE FINGERS

The last two phalanges are sometimes congenitally flexed owing to contraction of the digital fascia. The condition is sometimes hereditary, is often bilateral, and usually affects the little finger, but may also occur in the others. The treatment is similar to that of Dupuytren's contraction.

CLUB-HAND

Club-hand is a rare congenital deformity; it is usually associated with some other mal-development, or with absence or imperfect growth of the bones of the carpus or forearm; the radius is often absent, and in this case the ulnar is shortened and bowed with the concavity towards the radial side, so that the hand is deflected outwards.

Treatment consists in daily massage and manipulation, combined with division of all tendinous structures which may prevent the proper position of the hand being attained. In cases of absent radius it has been suggested to split the ulna longitudinally and thus form two bones.

Congenital Malformations of the Lower Urinary Tract, of the Genital Organs, and of the Rectum and Anus

Normal development.—At an early period of development the hind-gut, which is connected anteriorly with the allantois, and posteriorly by the post-anal gut with the neurenteric canal ends as a cul-de-sac, but at about the eighth week the gut and genito-urinary passage open on the surface by a common cloaca. Subsequently this cloaca is subdivided, and the communication between the allantois and the hind-gut is obliterated by the growth of a posterior and two lateral folds, which unite to form the perineum, and hence the gut remains posteriorly, and the urinary and genital passages open anteriorly, the opening being known as the uro-genital sinus (*fistulæ between rectum and bladder*). The hind-gut is completed by the proctodæum, which is a depression of the superficial structures; this gradually deepens until only a thin membranous septum separates the gut from the surface, the opening subsequently forming by absorption of this membranous partition (*imperforate anus*).

The post-anal gut is quite a temporary structure, and is early obliterated (*congenital sacral tumour*).

The lower end of the allantois and the Müllerian ducts open together into the uro-genital sinus, but become separated by a septum (*vesico-vaginal fistula*). The upper end of the allantois is obliterated, and remains only as a rounded cord (the urachus), passing upwards to the umbilicus from the bladder (*patent urachus, urachal cysts*).

The intermediate portion forms the bladder, and the lower part develops into the urethra of the female, but only forms the prostatic and membranous portions of that tube in the male, the penile portion being developed from the genital folds, as will be presently seen.

The Müllerian ducts remain separate above as the Fallopian tubes, but fuse below to form the uterus and vagina (*uterus bicornis, double vagina*); in the male these tubes are represented by the uterus masculinus. Thus at the uro-genital sinus, the urethra, and in the female the vagina opens, and most anteriorly is a small prominence (genital eminence), grooved on its under surface by the uro-genital furrow, and flanked on each side by a small fold (the genital fold). In the male the genital eminence forms the penis, while the edges of the genital furrow unite to form the penile urethra and corpus spongiosum, the union of its margins taking place from behind forwards (*hypospadias*); the genital folds unite in the middle line to form the scrotum (*cleft scrotum*). In the female the clitoris is developed from the genital eminence, while the sides of the genital furrow form the nymphæ, and the genital folds enlarge and give rise to the labia majora (*adherent labia*). The hymen appears at about the fifth month as a fold of mucous membrane. On each side of the female urethra two small orifices of Skene's tubes open—these are the lower ends of the Wolffian ducts, which in the male develop into the vasa deferentia and vesiculæ seminales.

NARROW MEATUS—IMPERFORATE URETHRA

The average circumference of the meatus in the male is about 25 mm., but it may be much larger, or so small as to cause actual obstruction to the flow of urine, coupled with painful micturition and signs of vesical irritation. A persistent gleet is sometimes traceable to the narrowness of the meatus. The meatus is usually quite small in cases of hypospadias. The defect is easily remedied

by passing a blunt-pointed knife into the urethra, and cutting the floor for a sufficient distance, the cut surfaces being kept apart for a few days by a small strip of oiled lint.

Occasionally the urethra is imperforate, and in such cases there may be no opening for the escape of urine and consequent secondary changes in the bladder or kidneys, or there may be an abnormal opening into the rectum or perineum. If the occlusion is due to a septum across the urethra, this must be broken down, but if the urethra is absent, a plastic operation, similar to that for hypospadias, must be performed.

EPISPADIAS

It sometimes happens that the penis is twisted, so that the urethra appears to occupy the dorsal surface, and if this is deficient the condition is known as epispadias. **Complete epispadias** is the usual form, and is associated with imperfect development of the pelvic girdle and extroversion of the bladder. The penis is stunted and rudimentary, being often only represented by an imperfect glans; the urethra opens at its root close to the pubes. For this condition there is no remedy. **Incomplete epispadias** may sometimes be benefited by a plastic operation similar to that practised for hypospadias.

HYPOSPADIAS

If the edges of the genital furrow fail to unite, the urethra is deficient, and opens on the under surface of the penis. The extent of this malformation varies, and three grades of hypospadias are recognised.

Varieties.—Hypospadias of the glans penis is a common condition; the floor of the urethra is deficient, and on the under surface of the glans is a distinct furrow. The prepuce is also deficient below, and surrounds the glans above and laterally like a cowl. This degree needs no treatment, but if the meatus is very small it should be incised.

Hypospadias of the penis.—The meatus opens on the floor somewhere between the glans and the scrotum, a groove on the under surface in front of the opening indicating the genital furrow. In front the corpus spongiosum is represented by a dense fibrous cord, which curves the stunted penis towards the scrotum, the curvature

being especially marked during erection. If the meatus is far back considerable inconvenience may result, and during connection the semen may not enter the vagina.

This defect may be remedied by operation.

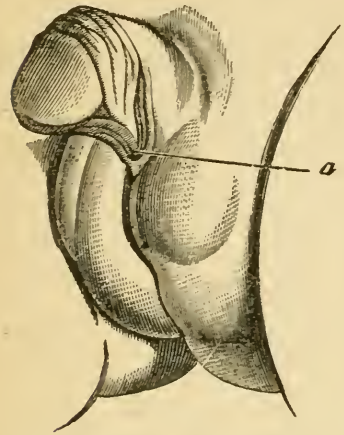

FIG. 108.—Peno-scrotal hypospadias. *a*, urethral orifice (Follin).

Complete hypospadias (H. perinealis).—In this condition the urethra opens on the perineum. The scrotum is cleft, the testes are often undescended, the penis is curved, short, stunted, and resembles an enlarged clitoris, so that on superficial examination the parts resemble the female genitals. The patient has to micturate like a woman, in the sitting position, and even if coitus can be performed the semen does not enter the vagina.

Hypospadias in women is very rare; the urethra opens into the vagina.

Operation for hypospadias. — It is advisable to operate when the patient is about ten or twelve years old, for he will then be able to understand the necessity for observing the directions given him, and the penis is sufficiently developed to offer a reasonable chance of success. These operations frequently fail owing to sloughing or inflammation due to the contact of the urine, or they may be only partially successful. It is first necessary to divide or dissect away the fibrous cord which keeps the penis in the curved condition, and the urethra may be formed at a subsequent operation. This may be effected by freshening the edges of the furrow, and uniting them over a catheter, but in the great majority of instances a small flap has to be raised from each side, and these are united with fine silk or chromic gut. For the details of the operation the reader is referred to a work on operative surgery.

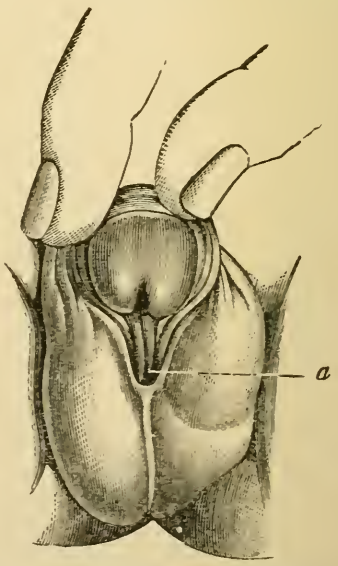

FIG. 109.—Peno-scrotal hypospadias. *a*, urethral orifice (Follin).

PATENT URACHUS

If the upper end of the allantois is unobliterated it opens at the umbilicus as a urinary fistula, which is usually quite small and may undergo spontaneous cure. The condition is very rare, and, especially if the opening be tolerably large, may be associated with umbilical hernia.

Sometimes the allantois is shut off below, so that there is no communication with the bladder, and the only evidence of its persistence is a narrow and perhaps very short sinus which may be the seat of suppuration.

If spontaneous cure does not occur, the sinus or fistula should be dissected out.

IMPERFECT OBLITERATION OF THE URACHUS WITH CYSTIC DILATATION—URACHAL OR ALLANTOIC CYSTS

The upper part of the allantois may be normally obliterated at the bladder and at the umbilicus, but not so as regards the intermediate portion, which may then become distended into a cyst or series of cysts. This condition is precisely like that which may occur in connection with the processus ad testem.

The cysts are situated between the abdominal wall and the parietal peritoneum. The cyst wall contains muscular fibres with fibrous tissue, and a lining of epithelium. The contents may be clear, and serous or mucoid. Such cysts may suppurate.

Treatment.—These cysts may be treated by excision or by free incision and drainage. The latter is the safer and more judicious course, since the peritoneal cavity is not opened, as would almost certainly be the case if any attempt were made to remove the cyst by dissection.

ECTOPIA VESICÆ

Extroversion of the bladder is due to failure of closure of the anterior abdominal wall with deficiency of the anterior wall of the bladder. The pubic arch is undeveloped and epispadias is associated; the testes are usually undescended and the scrotum is small. The posterior wall of the bladder projects as a red fungous mass in which the orifices of the ureters can be plainly seen. The constant dribbling of the urine renders the patient's life miserable, especially as puberty advances and the pubic hair

becomes encrusted with phosphates from the decomposing urine. The exposed mucous membrane not infrequently ulcerates and bleeds, but as age advances it becomes harder and its mucous character less marked. This deformity is much more common in males than females.

Operative treatment should usually be undertaken when the child is about five years old. The operations are tedious and complicated, and not infrequently fail completely or require repetition on account of sloughing. Trendelenburg recommends the restoration of the pelvic girdle before attempting to close the gap in the abdominal wall, and for this purpose exposes the sacro-iliac joints, divides the posterior ligaments, and then forces the pubic arch together and maintains the pelvis in this position by a properly applied girdle. This procedure tends to very much narrow the cleft, and renders the subsequent closure by flaps a less severe and more certain procedure.

CLEFT SCROTUM

This malformation is not uncommon, and may be associated with perineal hypospadias. The fault is due to failure of union of the genital folds. Each half of the scrotum contains a testicle if they have descended. No treatment is needed.

ADHERENT LABIA

If the genital folds unite in the female, the labia majora are united in the middle line. They should be incised or divided by the cautery along the raphé, and reunion must be prevented by the interposition of a piece of oiled lint during healing.

MALFORMATIONS OF THE RECTUM AND ANUS

From the general account given on p. 309 of the normal process of development the explanation of the malformations of the rectum will be easily understood.

Varieties—Anal stricture.—Sometimes the anal orifice is so small that it will only admit a fine probe. It should be enlarged by a median incision passing towards the coccyx and be kept open by bougies.

Imperforate rectum (Fig. 110).—This is the condition in

which the rectum and proctodæum are both developed, but have failed to unite owing to persistence of the septum, which must be broken down.

Imperforate anus (Fig. 111).—The rectum is normally developed, but the proctodæum is absent.

Absent rectum (Fig. 112).—The proctodæum may or may not

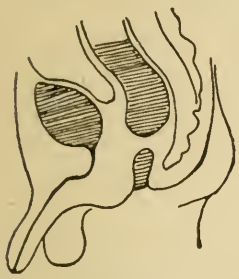

FIG. 110.—Imperforate rectum.

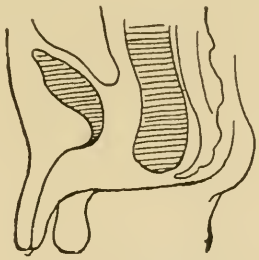

FIG. 111.—Imperforate anus.

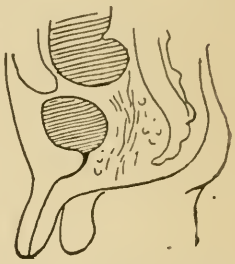

FIG. 112.—Absent rectum.

be developed, but the rectum is either quite absent or ends high up, its lower part being merely represented by a fibrous cord.

Atresia ani vesicalis (Fig. 113), **urethralis** (Fig. 114), **and vaginalis** (Fig. 115).—If the folds which should cut off the bowel from the urinary tract in front and should form the perineum are defective, one or other of these conditions will be present according

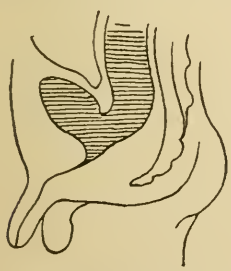

FIG. 113.
Atresia ani vesicalis.

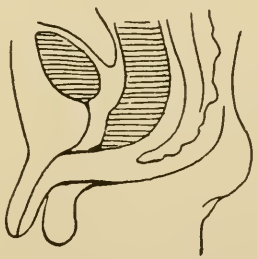

FIG. 114.
Atresia ani urethralis

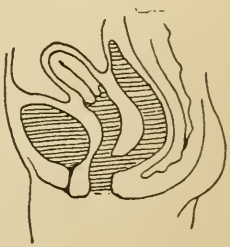

FIG. 115.
Atresia ani vaginalis.

to the sex of the patient and the length of the rectum; the longer the rectum the more likely is it to communicate with the urethra, and consequently the more easily can it be reached by operation.

Signs.—Whatever form of malformation is present the signs of obstruction soon become manifest, for even if there is an opening through the urethra or bladder, it is insufficient to permit of the free passage of meconium.

In cases of imperforate anus or rectum the distended bowel can easily be made out as it bulges into the perineum, especially when the child cries.

Treatment.—When the rectum is absent an attempt may be made to reach it by a careful dissection through the perineum, the coccyx being removed if necessary; the fibrous cord extending from the lower end of the bowel may be followed up to it if it can be defined. If the gut is found it should be drawn down and united to the skin, for unless this be done stricture is almost certain to occur. If the above operation fails, inguinal colotomy must be performed; this operation is also necessary in atresia ani vesicalis or urethralis.

Except in the very simplest forms of mal-development of the lower bowel the ultimate prognosis is very bad, for even if the child survive the operation he almost invariably dies in a few months.

CONGENITAL SACRAL TUMOUR

When the rectum opens on the surface by the formation of the proctodæum, the post-anal gut which communicates with the neurenteric canal should become obliterated. Sometimes remains of it persist and give rise to a dermoid or cystic tumour between the bowel and sacrum. Such a tumour may grow rapidly and attain a very large size so that the child looks a mere appendage of it. The tumour displaces the pelvic contents and grows downwards between the thighs, and also spreads upwards along the sacrum.

The cystic form of tumour is composed of numerous epithelium-lined cysts containing clear or mucoid fluid and frequently intracystic growths. Fatty and fibrous tissue is often a marked feature of the growth; cartilage, bone, and gland-tissue may also be met with.

These tumours should if possible be removed, but the operation is at all times dangerous in consequence of the attachments and pelvic prolongations of the growth.

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